

# ABNORMAL PSYCHOLOGY

FIRST AUSTRALASIAN EDITION | KRING



WILEY



# Abnormal psychology

1ST EDITION

Ann M. Kring

Michael Kyrios

Daniel Fassnacht

Amanda Lambros

Tijana Mihaljcic

Maree Teesson

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# CONTENTS

About the authors vii

## CHAPTER 1

### Introduction to abnormal psychology 1

#### Introduction 2

#### 1.1 The basic features of psychological disorder 3

What is psychological disorder? 3

#### 1.2 Stigma associated with psychological disorders and its historical context 6

Stigma and mental health 6

The historical context 8

#### 1.3 Early psychological approaches to psychopathology 10

#### 1.4 The genetic paradigm 16

Behaviour genetics 19

Molecular genetics 21

Gene–environment interactions 23

Evaluating the genetic paradigm 25

#### 1.5 The neuroscience paradigm 26

Neurons and neurotransmitters 26

Structure and function of the human brain 29

The neuroendocrine system 32

The immune system 33

Neuroscience approaches to treatment 35

Evaluating the neuroscience paradigm 38

#### 1.6 The cognitive–behavioural paradigm 39

Influences from behaviour therapy 39

Cognition 44

Cognitive–behavioural therapy 48

Beck's cognitive therapy 49

The Third Wave of cognitive therapies 52

Evaluating the cognitive–behavioural paradigm 53

#### 1.7 Factors that cut across the paradigms 53

Emotion and psychopathology 53

Sociocultural factors and psychopathology 55

Interpersonal factors and psychopathology 60

Attachment and the concept of the self in psychopathology 64

#### 1.8 Integration across multiple levels of analysis: the diathesis–stress integrative paradigm 66

## CHAPTER 2

### Diagnosis and assessment 88

#### Introduction 89

#### 2.1 Cornerstones of diagnosis and assessment 90

Reliability 90

Validity 91

#### 2.2 The diagnostic system of the American Psychiatric Association: DSM-5 94

Changes in DSM-5 97

Ethnic and cultural considerations in diagnosis 99

Specific criticisms of the DSM 104

#### 2.3 Psychological assessment 108

Clinical interviews 109

Assessment of stress 112

Personality tests 115

Intelligence tests 120

Behavioural and cognitive assessment 122

#### 2.4 Neurobiological assessment 126

Brain imaging: 'seeing' the brain 126

Neurotransmitter assessment 129

Neuropsychological assessment 130

Psychophysiological assessment 131

A cautionary note about neurobiological assessment 133

#### 2.5 Cultural and ethnic diversity and assessment 134

Cultural bias in assessment 134

Strategies for avoiding cultural bias in assessment 136

## CHAPTER 3

### Mood disorders 146

#### Introduction 147

#### 3.1 Clinical descriptions and epidemiology of mood disorders 147

Depressive disorders 147

Epidemiology and consequences of depressive disorders 152

Bipolar disorders 155

Epidemiology and consequences of bipolar disorders 159

Subtypes of depressive disorders and bipolar disorders 161

#### 3.2 Aetiology of mood disorders 162

Genetic factors 163

Neurotransmitters 164

Social factors in depression: childhood adversity, life events and interpersonal difficulties 169

Psychological factors in depression 170

Social and psychological factors in bipolar disorder 175

### 3.3 Treatment of mood disorders 176

Psychological treatment of depression 177

Psychological treatment of bipolar disorder 180

Biological treatment of mood disorders 181

Comparing treatments for major depressive disorder 184

A final note on treatment 186

### 3.4 Suicide 187

Epidemiology of suicide and suicide attempts 188

Risk factors for suicide 190

Preventing suicide 193

## CHAPTER 4

# Anxiety, obsessive-compulsive and trauma-related disorders 211

## Introduction 212

### 4.1 Clinical descriptions of the anxiety disorders 213

Specific phobias 214

Social anxiety disorder 216

Panic disorder 218

Agoraphobia 220

Generalised anxiety disorder 221

### 4.2 Comorbidity, gender and culture in anxiety disorders 222

Comorbidity in anxiety disorders 222

Gender and cultural factors in anxiety disorders 222

### 4.3 Aetiology of anxiety disorders 225

Common risk factors across the anxiety disorders 225

Aetiology of specific phobias 230

Aetiology of social anxiety disorder 232

Aetiology of panic disorder 233

Aetiology of agoraphobia 237

Aetiology of generalised anxiety disorder 237

### 4.4 Treatments of the anxiety disorders 238

Commonalities across psychological treatments 238

### 4.5 Obsessive-compulsive and related disorders 241

Clinical descriptions of the obsessive-compulsive and related disorders 242

### 4.6 Aetiology of the obsessive-compulsive and related disorders 249

Aetiology of obsessive-compulsive disorder 250

Aetiology of body dysmorphic disorder 251

Aetiology of hoarding disorder 251

### 4.7 Treatment of the obsessive-compulsive and related disorders 252

Medications 252

Psychological treatment 252

### 4.8 Post-traumatic stress disorder and acute stress disorder 255

Clinical description and epidemiology of post-traumatic stress disorder and acute stress disorder 256

Aetiology of post-traumatic stress disorder 258

### 4.9 Treatment of post-traumatic stress disorder and acute stress disorder 261

Medication treatment of PTSD 261

Psychological treatment of PTSD 262

## CHAPTER 5

# Dissociative disorders, and somatic symptom and related disorders 279

## Introduction 280

### 5.1 Dissociative disorders 281

Depersonalisation/derealisation disorder 283

Dissociative amnesia 285

Dissociative identity disorder 290

Other specified dissociative disorder 295

### 5.2 Somatic symptom and related disorders 296

Neurobiological and cognitive factors that increase awareness of and distress over somatic symptoms 299

Treatment of somatic symptom and related disorders 300

Somatic symptom disorder 300

Illness anxiety disorder 303

Conversion disorder 306

Factitious disorder 308

## CHAPTER 6

# Schizophrenia 319

## Introduction 320

### 6.1 Clinical descriptions of schizophrenia 321

Positive symptoms 321

Negative symptoms 326

Disorganised symptoms 327

Cognition 329

Anxiety and depression	330
Schizophrenia and the DSM-5	331
<b>6.2 Aetiology of schizophrenia</b>	<b>333</b>
Genetic factors	333
<b>6.3 The role of neurotransmitters</b>	<b>338</b>
Dopamine theory	338
Brain structure and function	339
Connectivity in the brain	343
<b>6.4 Environmental factors influencing the developing brain</b>	<b>344</b>
Psychological factors	345
Developmental factors	348
<b>6.5 Treatment of schizophrenia</b>	<b>349</b>
Medications	350
Psychological treatments	354

## CHAPTER 7

### Substance use disorders 375

<b>Introduction</b>	<b>376</b>
<b>7.1 Clinical descriptions, prevalence and effects of substance use disorders</b>	<b>376</b>
Alcohol use disorder	379
Tobacco use disorder	383
Cannabis	386
Opiates	390
Stimulants	393
Other illicit drugs	397
<b>7.2 Aetiology of substance use disorders</b>	<b>399</b>
Genetic factors	400
Neurobiological factors	400
Psychological factors	402
Sociocultural factors	404
<b>7.3 Treatment of substance use disorders</b>	<b>407</b>
Treatment of alcohol use disorder	407
Treatments for smoking	411
Treatment of drug use disorders	413
<b>7.4 Prevention of substance use disorders</b>	<b>418</b>

## CHAPTER 8

### Eating disorders 434

<b>Introduction</b>	<b>435</b>
<b>8.1 Clinical descriptions of eating disorders</b>	<b>436</b>
Anorexia nervosa	436
Bulimia nervosa	441
Binge-eating disorder	443
<b>8.2 Aetiology of eating disorders</b>	<b>448</b>
Genetic factors	448
Neurobiological factors	449
Cognitive-behavioural factors	451

Sociocultural factors	454
Other factors contributing to the aetiology of eating disorders	460

### 8.3 Treatment of eating disorders 463

Psychological treatment of anorexia nervosa	463
Psychological treatment of bulimia nervosa	464
Psychological treatment of binge-eating disorder	465
Medications	466
Preventive interventions for eating disorders	466

## CHAPTER 9

### Sexual disorders 480

<b>Introduction</b>	<b>481</b>
<b>9.1 Sexual norms and behaviour</b>	<b>481</b>
Gender and sexuality	485
The sexual response cycle	486
<b>9.2 Sexual dysfunctions</b>	<b>487</b>
Clinical descriptions of sexual dysfunctions	488
Aetiology of sexual dysfunctions	494
Treatments of sexual dysfunctions	497
<b>9.3 The paraphilic disorders</b>	<b>502</b>
Fetishistic disorder	504
Paedophilic disorder and incest	505
Voyeuristic disorder	508
Exhibitionistic disorder	508
Frotteuristic disorder	509
Sexual sadism and masochism disorders	509
Aetiology of the paraphilic disorders	512
Treatments for the paraphilic disorders	514

## CHAPTER 10

### Disorders of childhood 528

<b>Introduction</b>	<b>529</b>
<b>10.1 Classification and diagnosis of childhood disorders</b>	<b>530</b>
<b>10.2 Externalising disorders</b>	<b>532</b>
Attention-deficit hyperactivity disorder	532
Conduct disorder	540
<b>10.3 Depression and anxiety in children and adolescents</b>	<b>549</b>
Depression	549
Anxiety	553
<b>10.4 Specific learning disorder</b>	<b>561</b>
Clinical descriptions	561
Aetiology of specific learning disorder	562
Treatment of specific learning disorder	565
<b>10.5 Intellectual disability</b>	<b>565</b>
Diagnosis and assessment of intellectual disability	566

Aetiology of intellectual disability	567
Treatment of intellectual disability	569
<b>10.6 Autism spectrum disorder</b>	<b>572</b>
Clinical descriptions, prevalence and prognosis of autism spectrum disorder	572
Comorbidity and ASD	576
Prevalence of autism spectrum disorder	577
Prognosis for autism spectrum disorder	577
Aetiology of autism spectrum disorder	577
Treatment of autism spectrum disorder	580

## CHAPTER 11

### Late life and neurocognitive disorders 599

#### Introduction 600

#### 11.1 Ageing: issues and methods 600

Myths about late life	601
The problems experienced in late life	602
Research methods in the study of ageing	604

#### 11.2 Psychological disorders in late life 606

Estimating the prevalence of psychological disorders in late life	606
Methodological issues in estimating the prevalence of psychopathology	608

#### 11.3 Neurocognitive disorders in late life 609

Dementia	609
Delirium	626

## CHAPTER 12

### Personality and personality disorders 640

#### Introduction 641

#### 12.1 The DSM-5 approach to classification 642

Assessment of DSM-5 personality disorders	644
Problems with the DSM-5 approach to personality disorders	645
Alternative DSM-5 model for personality disorders	646

#### 12.2 Common risk factors across the personality disorders 648

#### 12.3 Clinical description and aetiology of the odd/eccentric cluster 650

Paranoid personality disorder	651
Schizoid personality disorder	651

Schizotypal personality disorder	652
----------------------------------	-----

#### 12.4 Clinical description and aetiology of the dramatic/erratic cluster 653

Antisocial personality disorder: clinical description	653
Psychopathy: clinical description	654
Borderline personality disorder	658
Histrionic personality disorder	662
Narcissistic personality disorder	662

#### 12.5 Clinical description and aetiology of the anxious/fearful cluster 666

Avoidant personality disorder	666
Dependent personality disorder	667
Obsessive-compulsive personality disorder	668

#### 12.6 Treatment of personality disorders 670

General approaches to the treatment of personality disorders	670
Treatment of schizotypal personality disorder and avoidant personality disorder	671
Treatment of borderline personality disorder	672

## CHAPTER 13

### Legal and ethical issues 684

#### Introduction 685

#### 13.1 Mental health and the criminal justice system 686

The insanity defence	687
Landmark cases and laws	688
The Mental Health Court	691
Magistrates Courts	693
Highly publicised trials and public perception	694

#### 13.2 Fitness to stand trial 694

Victims of unlawful acts	696
--------------------------	-----

#### 13.3 Civil commitment 697

Preventive detention and problems in the prediction of dangerousness	699
Deinstitutionalisation, civil liberties and mental health	709

#### 13.4 Ethical dilemmas in therapy and research 710

Ethical restraints on research	711
Informed consent	712
Confidentiality and ethical limits	714

# ABOUT THE AUTHORS

## **Ann M. Kring**

Ann M. Kring is Professor of Psychology at the University of California at Berkeley, where she is a former Director of the Clinical Science Program and Psychology Clinic. She received her BS from Ball State University and her MA and PhD from the State University of New York at Stony Brook. Her internship in clinical psychology was completed at Bellevue Hospital and Kirby Forensic Psychiatric Center, in New York. From 1991 to 1998, she taught at Vanderbilt University. She received a Distinguished Teaching Award from UC Berkeley in 2008. Ann is on the editorial board of *Schizophrenia Bulletin*, *Journal of Abnormal Psychology* and *Psychological Science in the Public Interest*, an Associate Editor for *Applied and Preventive Psychology* and a former Associate Editor for *Journal of Abnormal Psychology* and *Cognition and Emotion*. She is a former member of the Executive Board of the International Society for Research on Emotion.

Ann has won several awards, including a Young Investigator Award from the National Alliance for Research on Schizophrenia and Depression in 1997 and the Joseph Zubin Memorial Fund Award in recognition of her research in schizophrenia in 2006. In 2005, she was named a fellow of the Association for Psychological Science. Her research has been supported by grants from the Scottish Rite Schizophrenia Research program, the National Alliance for Research on Schizophrenia and Depression and the National Institute of Mental Health. She is a co-editor (with Denise Sloan) of the book *Emotion Regulation and Psychopathology* (Guilford Press) and is the author on more than 70 articles and chapters. Ann's current research focus is on emotion and psychopathology, with a specific interest in the emotional features of schizophrenia, assessing negative symptoms in schizophrenia and the linkage between cognition and emotion in schizophrenia.

## **Michael Kyrios**

Professor Michael Kyrios is Director of the Research School of Psychology at the Australian National University. His previous academic and clinical roles have included the Department of Psychiatry at the Royal Melbourne Hospital, the Departments of Psychiatry and Psychology at the University of Melbourne and the Brain and Psychological Sciences Research Centre at Swinburne University. Professor Kyrios's clinical and research interests focus on obsessive-compulsive spectrum disorders, including obsessive-compulsive disorder, body dysmorphic disorder, compulsive hoarding and buying, and body-focused repetitive behaviour disorders. He is particularly interested in evidence-based psychological treatments for these disorders, inclusive of digitally delivered treatments. Professor Kyrios has over 150 published journal articles and has given invited keynotes and workshops nationally and internationally. He has received over \$18 million in grant funding and is a consultant to government in his expert fields. Professor Kyrios was Scientific Chair for the 2010 International Congress of Applied Psychology and the 2016 World Congress of Behavioral and Cognitive Therapies. He served as President of the Australian Psychological Society from 2014–2016, and was elected a Fellow in 2007. He was also elected an Inaugural Fellow of the Australian Association of Cognitive Behaviour Therapy in 2016. Professor Kyrios received the 2011 Citation for Excellence from the American Psychological Association and the 2013 Ian M Campbell Memorial Prize in Clinical Psychology from the Australian Psychological Society.

## **Daniel Fassnacht**

Daniel Fassnacht is a Research Fellow at the Research School of Psychology at the Australian National University. In 2011 he was awarded a PhD in Psychology from the University of Tübingen, Germany, and the University of Minho, Portugal.

Daniel has extensive teaching experience in a wide range of psychology subjects in both undergraduate and postgraduate courses. He has held academic appointments in Germany, Portugal, Singapore

and Australia (University of Canberra and the Australian National University). Daniel holds a Graduate Certificate of Education (Tertiary Teaching) from James Cook University and has gained expertise in curriculum development and blended learning. Daniel's research spans from health to clinical psychology including depression, anxiety, obsessive-compulsive disorders, eating disorders and obesity. He has a special interest in the use of e-mental health applications to prevent and treat psychological disorders.

### **Amanda Lambros**

Amanda Lambros is a Clinical Fellow in the Faculty of Health Sciences at Curtin University and a practising clinical counsellor and supervisor. She has completed a BHSc in Health Sciences at the University of Western Ontario (2001), a Postgraduate Diploma of Ethics (2002), a Master of Forensic Sexology (2004) and a Master of Counselling (2014). Amanda coordinates and lectures in evidence-informed health practice to more than 3200 students annually. Amanda's private practice focuses on couples and grief and loss. Providing her clients with the most up-to-date and evidence-based care is imperative to her, and she has a strong focus on evidence-based practice, ethics and communication.

Amanda has extensive experience in teaching and speaking in Australia and overseas, in innovative teaching and evidence-based approaches and program development for health science courses. Before joining Curtin University, she held academic appointments at Curtin College, University of Southern Queensland and Griffith University. Amanda is a Certified Professional Speaker (CSP) who helps organisations understand 'evidence' and the power of communication to apply strategies to improve the underlying structure of their businesses. More recently, her speaking and consulting activities have been concentrated in the area of relationships, innovation, communication and engagement sectors all from an evidence-based perspective.

### **Tijana Mihaljcic**

Dr Tijana Mihaljcic is a published academic and practising clinical neuropsychologist. Tijana combines research and academic involvement with clinical practice on a weekly basis. She is passionate about using assessment to guide diagnosis, rehabilitation and management, ultimately leading to better client outcomes.

Dr Mihaljcic is a clinician in a Melbourne hospital and works with individuals diagnosed with a range of disorders including traumatic brain injury, stroke, neurodegenerative disorders, chronic pain and various psychiatric disorders. Her assessment findings are used to inform diagnosis and tailor interventions, delivering meaningful feedback to the client and their family.

Tijana's research interests include self-awareness, falls, elder financial abuse and memory rehabilitation post stroke. Her research has been published in peer-reviewed journals. Dr Mihaljcic also lectures at Monash University, where she is able to provide her students with up to date research and draw upon her experience as a clinician.

### **Maree Teesson**

Professor Maree Teesson is Director at the NHMRC Centre of Research Excellence in Mental Health and Substance Use (CREMS); NHMRC Principal Research Fellow at the National Drug and Alcohol Research Centre (NDARC); Professorial Fellow at the Black Dog Institute, UNSW; Fellow at the Australian Academy of Health and Medical Sciences; and Fellow at the Australian Academy of Social Sciences.

Maree's vision is to build the world's leading dedicated translational research program for the prevention and treatment of comorbid mental health and substance abuse. She seeks to increase our understanding of drug and alcohol and mental disorders, prevent these where possible and improve treatment responses. Maree has made a substantial contribution to medical research with over 280 research articles, reviews, book chapters and books. The innovation of her research has been recognised through leadership of over 90 grants totalling over \$44 million.

### **Mandy Matthewson**

Dr Mandy Matthewson is a clinical psychologist and has been practising since 2005. She currently practises psychology in private practice. Dr Matthewson is also a lecturer in psychology at the University of

Tasmania and has held this position since 2007. Dr Matthewson has extensive knowledge and expertise in teaching a variety of core and applied areas of psychology such as psychological assessment, abnormal psychology and clinical psychology practice. Her areas of research interest include family relationships, developmental psychology and the impact of family conflict on children's psychosocial development. Dr Matthewson is a member of the Australian Psychological Society and College of Clinical Psychologists. She is also an associate editor of *Clinical Psychologist*.

### **Emma Morton**

Emma Morton is a PhD candidate (Clinical Psychology) and provisional psychologist based at Swinburne University of Technology. Emma has taught undergraduate psychology at Swinburne University since 2017, and has also presented workshops on therapeutic tools and techniques for working with people with bipolar disorder.

Emma's clinical and research interest is in improving the lives of those with severe mental illnesses, particularly bipolar spectrum disorders. Emma has interned as a provisional psychologist at St Vincent's Hospital Adult Inpatient Mental Health Unit and Orygen Youth Health Early Psychosis Prevention and Intervention Centre. Her research to date has focused on understanding, measuring and improving quality of life in bipolar disorder, with a particular emphasis on empowering consumers by representing their preferences and perspectives in research. Emma is a trainee member of the collaborative research team to study psychosocial issues in bipolar disorder, CREST.BD, and is a contributor on their bipolar blog.

### **Mary-Anne Kate**

Mary-Anne Kate is a PhD Candidate (Psychology) at the University of New England. Her doctoral research examines how childhood maltreatment and negative parent-child dynamics relate to dissociation and dissociative disorders in adulthood. Mary-Anne is a member of the International Society for the Study of Trauma and Dissociation's Scientific Committee. Her professional background is in the development of national and European Union policies and practices to improve quality of life outcomes, including mental health, for refugees and other minority groups. Mary-Anne was awarded a Master's degree in social policy with distinction from the University of Edinburgh.

### **Anthony Harris**

Dr Anthony Harris is an Associate Professor in the Discipline of Psychiatry at the University of Sydney and is the Clinical Director of the Brain Dynamics Centre, Westmead Institute for Medical Research. He works in the Prevention Early Intervention and Recovery Service in Parramatta seeing young people with a range of severe mental illnesses. He is the current President of the One Door Mental Health and is on the Board of the Mental Illness Fellowship of Australia. He is chair of the Schizophrenia Research Council of Neuroscience Research Australia.

### **Zahra Izadikhah**

Dr Zahra Izadikhah is a lecturer at the University of Southern Queensland. She is a registered psychologist and a member of the Australian Psychological Society. She received her PhD in psychology from the University of Queensland in 2009. She has also completed a Bachelor and a Masters of Clinical Psychology. Zahra has taught postgraduate, honours and undergraduate programs and has taught courses in the areas of clinical psychology, psychotherapy and counselling, health, and personality theories. Her main research interests are body and mind interaction, and pain and somatisation and psychosomatic disorders. She investigates the structure and dynamics of personality, dynamics of the unconscious, trauma and abuse, and emotion regulation, as well as conscious and unconscious processes/mechanisms for change in psychotherapy. She has had professional training in intensive short-term dynamic psychotherapy and over 14 years of experience of clinical practice across various organisations including private clinics and universities.

### **Adina Piovesana**

Adina Piovesana is a lecturer of psychology in the Faculty of Psychology and Counselling at the University Southern Queensland, a position she has held since graduating with her doctorate in 2014.

Adina has experience teaching ethics and psychological assessment to undergraduate psychology students and has a developing research profile in the fields of psychometrics and psychological assessment.

### **Lynda Crowley-Cyr**

Lynda Crowley-Cyr is Associate Professor of Law in the School of Law and Justice at the University of Southern Queensland, a position she has held since 2006. Prior to this, she was a Senior Lecturer in the Faculty of Law at James Cook University since 1992.

Lynda has extensive experience in teaching health law in Australia and overseas, to both law students and students in various health care disciplines, and in course program development and management. Before joining the University of Southern Queensland, she was a presiding member of the Mental Health Review Tribunal in Queensland and engaged in consulting activities at hospitals providing lectures on various health law topics.

Prior to her academic career, Lynda worked in several law firms representing clients mainly in the areas of criminal law and family law. Prior to that, she was a nurse and then a homemaker while completing her law studies. Lynda has published extensively in the health and mental health law literature and her current research interests are in the fields of negligence and mental health, health security and public authority liability.

### **Andrew Baillie**

Andrew Baillie is Professor at the University of Sydney, Faculty of Health Sciences and a member of the Centre of Emotional Health at Macquarie University. He is a registered psychologist with an endorsement in clinical psychology and a board approved clinical supervisor. He also holds an honorary appointment as a clinical psychologist at Royal Prince Alfred Hospital, Sydney. Andrew has published on anxiety disorders and comorbidity with alcohol use disorders from an assessment, treatment and epidemiological perspective.

### **Louise Mewton**

Dr Louise Mewton is a Senior Research Fellow at the National Drug and Alcohol Research Centre (NDARC), University of New South Wales. Louise's research focuses on the application of innovative methods, techniques and technologies to further our understanding of the epidemiology, classification and prevention of substance misuse and mental illness during the critical adolescent period. Her program of research makes links across epidemiology, information technology, neuropsychiatry and prevention research, and reflects global research priorities.

Louise has published extensively on the epidemiology and classification of substance use disorders, and focuses on the application of this knowledge to inform preventative interventions aimed at reducing the prevalence of these disorders in the general population.

### **Luke Johnson**

Luke Johnson is a lecturer and researcher at the Queensland University of Technology in the School of Psychology and Counselling. His research investigates fundamental questions in the neurobiology of fear memory and stress, with the aim of providing vital basic knowledge into the way the brain encodes normal and pathological fear memories.

### **Warwick Middleton**

Warwick Middleton MB BS, FRANZCP, MD., holds appointments as Adjunct Professor, School of Public Health, La Trobe University; School of Behavioral, Cognitive & Social Sciences, University of New England; Department of Psychology, University of Canterbury; and Associate Professor in Psychiatry, University of Queensland. He has made substantial and ongoing contributions to the bereavement and trauma literatures and was co-author of the first published series in Australian scientific literature detailing the abuse histories and clinical phenomenology of patients fulfilling diagnostic criteria for Dissociative Identity Disorder. He chairs The Cannan Institute as well as its research and conference organising committees. In 1996 he was the principal architect in establishing Australia's first dedicated unit

treating dissociative disorders (the Trauma and Dissociation Unit, Belmont Hospital) and he continues as its Director. He has been in full time private practice since 1995.

Professor Middleton has had substantive ongoing involvement with research, writing, reviewing, teaching (including workshops and seminar presentations), conference convening, forensic reporting and supervision of health and research professionals. He is a member of the International Advisory Board of the Australian and New Zealand Journal of Psychiatry and is a member of the Editorial Board of the Journal of Trauma and Dissociation. In 2008 he was elected a Fellow of the International Society for the Study of Trauma and Dissociation (ISSTD). Professor Middleton served on the ISSTD committee revising the Treatment Guidelines for Dissociative Identity Disorder (2011), is vice-chair of the ISSTD Research Committee and he co-chairs the ISSTD Membership Committee. A particular area of ongoing research interest is ongoing incestuous abuse during adulthood. In 2013 this work was recognised with the ISSTD Morton Prince Award for scientific achievement. He was a joint-recipient of the 2014 ISSTD Pierre Janet Award for Writing, in 2015 the ISSTD President's Award, as well as the RANZCP 2015 Joan Lawrence Queensland Meritorious Service Award, and in 2017 the ISSTD Distinguished Achievement Award. He is the Immediate Past President of the ISSTD and he chaired an editorial grouping that put together a Special (double) Issue of the Journal of Trauma and Dissociation ('The Abused and the Abuser: Victim–Perpetrator Dynamics'), published in 2017.



## CHAPTER 1

# Introduction to abnormal psychology

### LEARNING OUTCOMES

---

After studying this chapter, you should be able to:

- 1.1** describe the basic features of psychological disorder
  - 1.2** understand stigma associated with psychological disorders and its historical context
  - 1.3** understand early psychological approaches to psychopathology
  - 1.4** describe the essentials of the genetic paradigm
  - 1.5** describe the essentials of the neuroscience paradigm
  - 1.6** describe the essentials of the cognitive–behavioural paradigm
  - 1.7** understand emotion, culture, ethnicity and interpersonal factors in the study and treatment of psychopathology
  - 1.8** recognise the importance of integration across multiple levels of analysis: the diathesis–stress integrative paradigm.
-

## OPENING SCENARIO

Emma and Ahmed, both in their mid 30s, were attending relationship counselling following a period of conflict in their marriage that had further impaired their already significant mental health problems. They had been married for around 5 years and, while they had always quarrelled, the decision as to whether to start a family led to an exacerbation of their difficulties. Emma and Ahmed come from very different cultural and experiential backgrounds, and this has contributed to disagreements about the roles each should take in their household.

In the past, Emma and Ahmed had experienced periods of significant individual trauma and distress. They met in their mid-20s at an outpatient psychiatric day-program they were attending, Emma for schizophrenia and Ahmed for post-traumatic stress disorder. Despite their very different backgrounds, they shared much in terms of their mental health experiences and the stigma they have endured.

Emma had a mixed Indigenous Australian and Irish Catholic family background and her father had been in a psychiatric hospital for over 10 years. She was always socially anxious and preferred to be alone. She had few friends and had been bullied and ostracised at school, while her teachers regarded her as 'unusual'. Emma's mother had suffered from depression but raised four children as best she could. Emma was not successful academically, and psychological assessments concluded she had numerous cognitive deficits. During secondary school, Emma started hearing voices, something she found enormously distressing and debilitating. Her treatment comprised hospitalisation, antipsychotic medications, psychological treatments and various social programs. Although Emma still occasionally hears voices, they no longer distress her and she has been able to maintain independence and part-time work as an administrative assistant.

Ahmed was born in the Middle East and, despite a loving family and relatively stable early childhood, was traumatised during the war when his father died in a bomb attack as the family fled to a foreign refugee camp. Ahmed has suffered from flashbacks and emotional turmoil since then, which have waxed and waned over the years. He also developed suicidal thoughts and felt guilty about surviving the bombing. When he was in the refugee camp he stopped communicating with anyone, and medical assessments concluded he might be intellectually disabled. Ahmed immigrated to Australia during his adolescence with his surviving family where he was able to successfully complete his education, although at times he was suicidal and attended psychiatric services. A **diagnosis** of post-traumatic stress disorder led to appropriate treatment inclusive of medications, cognitive-behavioural therapy and social services. Ahmed still experiences periods of depression and occasional panic attacks, but he has maintained a successful career as a public servant.

Relationship counselling helped Emma and Ahmed gain insight and resolve their ambivalence about having a child, thus improving their mental health and sense of wellbeing. Emma acknowledged she had been anxious about her family history of mental illness and an expectation that her offspring would be encumbered by her 'genetics', while Ahmed was concerned about his perceived cultural expectations to be a father. Given their ongoing mental health challenges, they were pessimistic about being able to cope with future parenthood.

### QUESTIONS

1. What factors led to Emma and Ahmed each having developed a mental health disorder?
2. Are the current difficulties Emma and Ahmed are experiencing normal or due to their mental health problems?
3. What factors might influence the reticence of Emma and Ahmed to be open about their problems?
4. Were the assessments administered to Emma and Ahmed adequate, given their backgrounds and experiences?
5. How important is a diagnosis in determining appropriate treatment?

## Introduction

The term **psychopathology** describes the scientific study of abnormal behaviour in general, of specific psychological disorders (also commonly referred to as 'mental disorders') and the characteristics of mental health. Essential to the field of psychopathology is the definition of *disorder*. Throughout the

ages, several waves of historical, social, religious, cultural and scientific influences have been used to define, understand, classify and manage abnormal behaviour. However, many of these attempts have led to misunderstanding, stigma and inappropriate or even inhumane management of affected individuals. Nonetheless, since the introduction of contemporary scientific methods in studying normality and abnormality, we have seen improved understanding, greater social acceptance and enhanced treatment options for people with mental health problems that ultimately have achieved greater wellbeing for individuals and communities.

Given the nature of human beings and our history, it is not surprising that social, religious and cultural factors have played an important role in understanding psychological disorder. However, the scientific method has enabled the identification of biological and psychological phenomena that have helped further define and differentiate normality and abnormality.

## 1.1 The basic features of psychological disorder

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**LEARNING OUTCOME 1.1** Describe the basic features of psychological disorder.

### What is psychological disorder?

Contemporary definitions of **psychological disorder** contain several features, with no single aspect being sufficient to categorically differentiate normality from abnormality. These include the following.

- *The impact on the life of affected individuals of associated clinical phenomena such as behaviours, thoughts and emotions.* Personal distress and disability or interference with one's wellbeing, social adjustment and occupational or academic and personal life are important indicators of the negative impact associated with psychological disorder.
  - A person's behaviour may be classified as disordered if it causes him or her great distress. Both Emma and Ahmed from the opening scenario had experienced great distress as a result of their **symptoms** (e.g., from the 'voices' for Emma and the 'flashbacks' for Ahmed). Personal distress characterises many of the forms of psychological disorder considered in this text — for example, people experiencing **anxiety disorders** and **depression** suffer greatly. But not all psychological disorders cause distress. For example, an individual with the antisocial type of personality disorder may treat others coldheartedly and violate the law without experiencing any guilt, remorse, **anxiety** or other types of distress. And not all behaviour that causes distress is disordered — for example, the distress of hunger due to religious fasting or the pain of childbirth.
  - Disability — that is, impairment in some important area of life (e.g., work, study or personal relationships) — can also characterise psychological disorder. While distress can lead to disability, it is not always necessary. For instance, the features of antisocial personality disorder may not cause distress, but they do interfere with social and occupational functioning. Furthermore, substance use disorders are defined in part by the social or occupational disability (e.g., serious arguments with one's spouse or poor work performance) created by substance abuse. Being rejected by peers, as Emma was, is also an example of this characteristic. **Phobias** can produce both distress and disability — for example, if a severe fear of flying prevents someone from working effectively in a multinational company that requires frequent travel. Like distress, however, disability alone cannot be used to define psychological disorder because some, but not all, disorders involve disability. For example, the disorder bulimia nervosa involves binge eating and compensatory purging (e.g., vomiting) in an attempt to control weight gain, but it does not necessarily involve disability. Many people with bulimia lead lives without significant impairment, while bingeing and purging in private. Other characteristics that might, in some circumstances, be considered disabilities — such as being blind but wanting to become a professional race car driver — do not fall within the domain of psychopathology. We do not have a rule that tells us which disabilities belong in our domain of study and which do not.
- *The degree of threat or actual danger that specific behaviours bear on affected individuals or to the community more generally.* In an influential and widely discussed paper, Wakefield (1992) proposed

that psychological disorders could be defined as **harmful dysfunction**. This definition has two parts: a value judgement ('harmful') and an objective, scientific component — the 'dysfunction'. A judgement that a behaviour is harmful requires some standard, and this standard is likely to depend on social norms and values. While many people may regard the 'harm' feature as an obvious characteristic of abnormality, it is one that can be difficult to recognise, detect and document reliably. One difficulty is that many internal mechanisms involved in psychological disorders are undetermined; thus, we cannot say exactly what may not be functioning properly. Wakefield (1999) has tried to meet this objection by, in part, referring to plausible dysfunctions rather than proven ones. In the case of Ahmed, for example, flashbacks could be construed plausibly as a failure of the mind to 'turn off' unwanted images or memories rather than 'dangerous' or 'harmful' per se. However, his suicidal thoughts are definitely of concern and can be considered as potentially dangerous to his wellbeing. Nevertheless, we have a situation in which we judge a behaviour or set of behaviours to be harmful and then decide that the behaviour represents a psychological disorder because we believe it is caused by a dysfunction of some unknown internal mechanism. Clearly, like the other definitions of psychological disorder, Wakefield's concept of harmful dysfunction has its limitations.

- *The frequency of behaviours.* While infrequent behaviours or responses to life situations may be indicative of abnormality, they could also be adaptive. For instance, being a genius may be infrequent but is not considered abnormal. On the other hand, high frequency behaviours can impact negatively on wellbeing. For instance, 2007 national survey data indicates that 45.5 percent of Australians will present with a mental health disorder at some point in their lives (Slade, Johnston, Oakley Browne, Andrews, & Whiteford, 2009). One important feature of a statistical approach to defining abnormality is that it highlights the dimensional nature of psychological characteristics and provides the impetus to focus on the potential adaptive or evolutionary advantages of such characteristics. For example, while high states of anxiety can be disabling, some anxiety is actually highly motivating and adaptive. Consider the observation that students tend to study more when they are somewhat concerned about an upcoming exam; however, too much anxiety can impede one's ability to learn.
- *Social norms and expectations.* In the realm of behaviour, social norms are widely held standards (beliefs and attitudes) that people use consciously or intuitively to make judgements about where behaviours are situated on such scales as good–bad, right–wrong, justified–unjustified and acceptable–unacceptable. Deviance from socially acceptable behaviour has long been used to determine abnormality but this feature is fraught with difficulties and characterised by opposition from many researchers. For example, the behaviours and motivations of people imprisoned for sexual offences against children clearly violate social norms. Yet this way of defining psychological disorder is both too broad and too narrow. For example, it is too broad in that criminals violate social norms but are not always studied within the domain of psychopathology; it is too narrow in that those who have similar thoughts but do not act on them because of high anxiety regarding the consequences typically do not violate social norms. Also, of course, social norms vary a great deal across cultures and ethnic groups, so behaviour that clearly violates a social norm in one group may not do so at all in another. For example, in some cultures but not in others, directly disagreeing with someone violates a social norm. Throughout this text, we will address this important issue of cultural and ethnic diversity as it applies to the descriptions, causes and treatments of psychological disorders. Moreover, the violation of social norms is not always indicative of abnormality but can be an integral part of positive community evolution, as was seen in the feminist movement, which opposed gender biases in role expectations. Another specific example of how changing social norms impact directly on the definition of disorder can be seen in the way that homosexuality was dealt with by the mental health professions. While once considered a disorder, homosexuality is now regarded as a normal variant, although how this change manifests varies depending on culture, religion and socioeconomic and educational contexts. Furthermore, although all are indicative of nonconformity, there are important differences in the impact of quirkiness and eccentricity versus serious criminal acts like paedophilia, the latter being

more clearly indicative of pathology. Although nonconformity may impact on social adjustment, psychopathology reflects a broader range of criteria. Hence, contextual deviance on its own is not necessarily a reliable or valid indicator of pathology.

- *Biological differences, including abnormal physiological features (e.g., brain structures and functions) and genetic abnormalities.* Such abnormalities may manifest in abnormal reactions, behaviour, emotions and cognitions, which will impact on how individuals can adjust to the challenges of life. We will cover biological influences on psychopathology in more detail in following sections, although it is important to note that individuals with even the same psychological disorder vary enormously in their biological features.

No single characteristic can fully define the concept of abnormality and each attribute provides advantages and disadvantages in defining pathology. Consequently, psychological disorder is usually determined based on the presence of several characteristics at one time. Several formal frameworks are used to determine abnormality and to diagnose disorders. The most commonly used frameworks internationally are as follows.

- The International Statistical Classification of Diseases and Related Health Problems (ICD) is a medical classification list by the World Health Organization (WHO). The eleventh revision of the ICD was initiated by WHO in 2007 and is expected to be complete by 2018. A beta draft is currently available (see <http://apps.who.int/classifications/icd11/browse/f/en>).
- The fifth edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* of the American Psychiatric Association (APA) was released in May 2013. It has been criticised by various associations, such as the Australian Psychological Society and the British Psychological Society. Nonetheless, the DSM-5 asserts a number of characteristics of psychological disorder, including:
  - the disorder occurs within the individual
  - it involves clinically significant difficulties in thinking, feeling or behaving
  - it usually involves personal distress of some sort, such as in social relationships or occupational functioning
  - it involves dysfunction in psychological, developmental and/or neurobiological processes that support mental functioning
  - it is not a culturally specific reaction to an event (e.g., death of a loved one)
  - it is not primarily a result of social deviance or conflict with society.

Classification systems such as the DSM-5 have been criticised due to the ‘in’ or ‘out’ nature of diagnosis. They assume that one either has or does not have a disorder, rather than considering a dimensional framework. Symptoms associated with all disorders can be seen throughout the population and are not unique to specific disorders. For instance, mood problems, intrusive thoughts, and unusual beliefs and thinking are not seen specifically in depression, obsessive-compulsive disorder and schizophrenia, respectively. Classification systems also tend to ‘medicalise’ problems of daily living. Depression and anxiety are a part of life and need not be considered abnormal conditions. A range of other criticisms can be identified with respect to diagnostic systems in general and the DSM specifically (Murray, 2012).

It is clear that we face enormous challenges in remaining objective when trying to understand and study psychopathology scientifically. Above, we have already introduced the idea that culture and context impact on how we understand and define psychological disorder. Science is a human enterprise that is bound by scientists’ human limitations; it is also bound by the current state of scientific knowledge and the scientific method itself. We are bound by the scientific method’s adherence to hypothesis testing, which fundamentally limits us to asking questions and investigating phenomena that do not go much beyond what we already understand. Furthermore, while every effort should be made to study psychopathology according to scientific principles, we need to understand that science is not a completely objective and certain enterprise. Rather, as suggested by Thomas Kuhn (1970), a major science philosopher, subjective factors as well as our human limitations impact on the conduct of scientific inquiry.

Central to scientific activity, in Kuhn's view, is the notion of a **paradigm**, a conceptual framework or approach within which a scientist works — that is, a set of basic assumptions, a general perspective, that defines how to conceptualise and study a subject, how to gather and interpret relevant data, even how to think about a particular subject. A paradigm has profound implications for how scientists and clinicians operate at any given time. Paradigms specify what problems scientists will investigate and how they will go about the investigation. O'Donohue (1993) has criticised Kuhn's use of the concept of paradigm, noting that he was inconsistent in its definition. The complexities of this argument are beyond the scope of this text. Suffice to say that we find it useful to organise our thinking about psychological disorders around the paradigm concept. We use the term to refer to the general perspectives that constrain the way scientists collect and interpret information in their efforts to understand the world.

In this chapter we consider current paradigms of psychopathology and treatment. Consistent with a broader **biopsychosocial framework** that highlights the importance of biological, psychological and social factors that impact on our understanding of normal and abnormal behaviour, we present three paradigms that are particularly influential in the study and treatment of psychopathology: genetic, neuroscience and cognitive-behavioural. We also consider the important role of emotion and sociocultural factors in psychopathology. These factors cut across all the paradigms and are significant in terms of the description, causes and treatments of all the disorders we will discuss in this text.

Current thinking about psychopathology is multifaceted. The work of clinicians and researchers is informed by an awareness of the strengths and limitations of all the paradigms they use. For this reason, current views of psychopathology and its treatment typically integrate several paradigms. At the end of this chapter we describe another paradigm — diathesis-stress — that provides a major basis for an integrative approach.

For researchers and clinicians, the choice of a paradigm has important consequences for the way in which they define, investigate and treat psychopathology. Our discussion of paradigms will lay the groundwork for the topics covered in the rest of the text. We note at the outset that no one paradigm offers the 'only' or the 'best' conceptualisation of psychopathology. Rather, for most disorders, each paradigm offers some important information with respect to **aetiology** and treatment, but provides only part of the picture. In a sense, the major paradigms illustrate *general* influences that act on behaviour but each disorder is also associated with *specific* factors that will ultimately help clinicians understand affected individuals.

Before going on to discuss the various major paradigms used to understand psychopathology, it is important to understand one of the greatest challenges to people experiencing mental health difficulties, that of stigma.

## 1.2 Stigma associated with psychological disorders and its historical context

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**LEARNING OUTCOME 1.2** Understand stigma associated with psychological disorders and its historical context.

**Stigma** refers to the destructive beliefs and attitudes held by a society that are ascribed to groups considered different in some manner, such as people with psychological disorders.

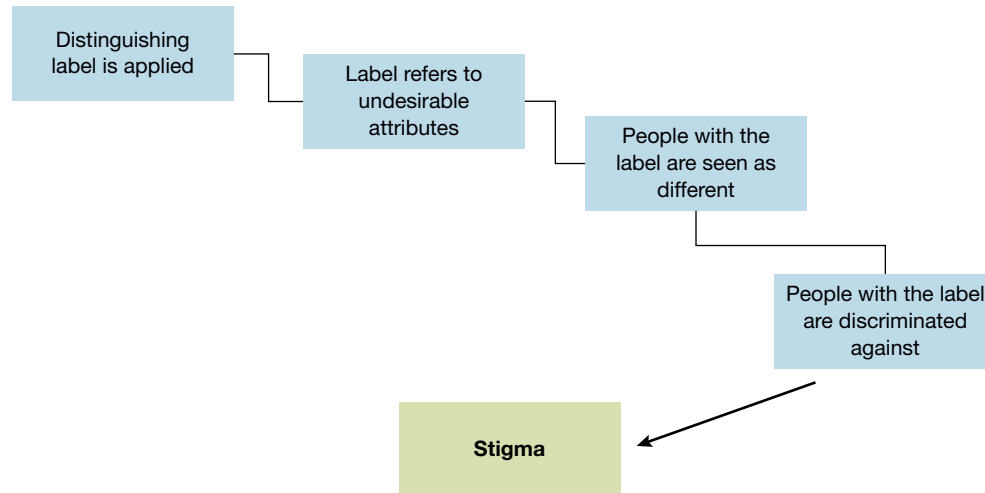
### Stigma and mental health

Stigma encompasses: (a) ignorance or limitations in *knowledge* about mental health; (b) prejudice or negative *attitudes* to mental illness; and (c) unhelpful or *discriminatory responses* to people with mental health problems (see Thornicroft, Rose, Kassam, & Sartorius, 2007). More specifically, stigma has four characteristics (see figure 1.1).

1. A label is applied to a group of people that distinguishes them from others (e.g., 'crazy').
2. The label is linked to deviant or undesirable attributes by society (e.g., 'crazy people are dangerous').

3. People with the label are seen as essentially different from those without the label, contributing to an 'us' versus 'them' mentality (e.g., 'we are not like those crazy people').
4. People with the label are discriminated against unfairly (e.g., 'a clinic for crazy people can't be built in our neighbourhood').

**FIGURE 1.1** The four characteristics of stigma



However, while most stigma research focuses on the attitude of others towards people with mental health problems, termed *personal or public stigma*, there are also other aspects of stigma. Many people with mental illnesses can have attitudes about themselves that are harsh or discriminatory ('I am no good', 'I can never function properly', 'I shouldn't bother getting treatment'). These forms of stigma are commonly referred to as *self-stigma* and refer to the extent to which one accepts or internalises the negative attitudes of others towards one's self. However, stigma can also concern how one perceives the attitudes of others towards mental illness (*perceived stigma*). The case of Emma illustrates the effects of stigma. Emma was bullied and ostracised at school, while she also held on to discriminatory fears about having a child who might be 'burdened' by a mental health illness. Carr and Halpin (2002) describe stigma as follows.

The term 'stigma' means a mark or sign of disgrace or discredit and 'to stigmatise' means to regard a person as unworthy or disgraceful. The consequences of being regarded in such a way include shame, humiliation, ostracism and despair. The burden of mental illness is thus made even heavier, not only by the direct effects of stigmatisation but by the profound injustice in being thus regarded. (p. 1)

Stigma appears to affect some people or groups more than others and can vary for different cultures, religions, social and educational groups and ages. Furthermore, people with different diagnoses vary in the degree of discrimination they experience. For instance, an Australian survey by the Wesley Mission (2007) found that depression and anxiety were associated with relatively generous and compassionate community attitudes but differed in terms of how well they were understood — public awareness campaigns within Australia had probably rendered depression to be a better accepted and understood mental health problem. **Schizophrenia** and bipolar disorder, on the other hand, were poorly understood and associated with negative community attitudes.

Stigma can be influenced by a number of factors, including culture, education and personal experiences, but 'mental health literacy' (i.e., knowledge and awareness about mental illness, symptoms, causal factors and treatment) can differentially affect personal, perceived and self-stigma. The effects of stigma can be devastating. Stigma has a negative influence on self-esteem and help seeking, thus impacting on **prognosis**. It diminishes opportunities for socialisation and employment, thus promoting

social isolation. Discrimination is a significant obstacle as it impacts on affected individuals' experiences with their families and the broader community as well as the quality of their experiences with services. Nonetheless, in order to better understand the origins of stigma, we need to better understand its historical stigma.

## The historical context

Stigma has long been associated with psychopathology, with much of the stigma ascribed to historical accounts relating to the aetiology of mental illness. Early concepts of psychological disorders included **demonology** (i.e., the belief that odd behaviour was caused by possession by demons or evil spirits), leading to treatment via unscientific and often inhumane methods (e.g., **exorcism**, elaborate rites of prayer, being forced to drink terrible-tasting brews, and even torture such as flogging and starvation to render the body uninhabitable by 'devils').

In the fifth century BC, Hippocrates (c.460–c.377 BC), the father of modern medicine, separated medicine from religion, magic and superstition and expounded the notion that mental disturbances were illnesses with natural causes like other, more common maladies, such as colds and constipation. As such, he rejected the prevailing belief that the gods sent mental disturbances as punishment and insisted instead that such maladies were treatable. Hippocrates regarded the brain as the organ of consciousness, intellectual life and emotion; thus, he thought that disordered thinking and behaviour were indications of some kind of brain pathology. Hippocrates is often considered one of the earliest proponents of the notion that something wrong with the brain disturbs thought and action.

Through Hippocrates's teachings, the phenomena associated with psychological disorders became more clearly the province of physicians rather than priests. The treatments he suggested were quite different from exorcism. Because Hippocrates believed in natural rather than supernatural causes, he depended on his own keen observations and made valuable contributions as a clinician. He also left behind remarkably detailed records clearly describing many of the symptoms now recognised in seizure disorders, alcohol dependence, stroke and paranoia.

Hippocrates's ideas, of course, did not withstand later scientific scrutiny. However, his basic premise — that human behaviour is markedly affected by bodily structures or substances and that odd behaviour is produced by some kind of physical imbalance or even damage — did foreshadow aspects of contemporary thought. In the next seven centuries, Hippocrates's naturalistic approach to disease and disorder was generally accepted.

However, following the death of Galen (AD 130–200), the second-century Greek who is regarded as the last great physician of the classical era, the so-called Dark Ages in Western European medicine and in the treatment and investigation of psychological disorders commenced. The Church gained in influence and Christian monasteries, through their missionary and educational work, replaced physicians as healers and as authorities on psychological disorder. Thus began a return to beliefs about the supernatural causes of psychological disorders. During the Dark Ages, some people with psychological disorders were cared for in monasteries, but many simply roamed the countryside. Some were persecuted as witches, although later analyses indicated that many of the people accused of being witches were not mentally ill.

Nonetheless, from the thirteenth century on, as the cities of Europe grew larger, hospitals began to come under secular jurisdiction. Municipal authorities, gaining in power, tended to supplement or take over some of the activities of the Church, one of these being the care of people who were mentally ill. The foundation deed for the Holy Trinity Hospital in Salisbury, England, dating from the mid-fourteenth century, specified the purposes of the hospital, one of which was that the 'mad are kept safe until they are restored of reason'. English laws during this period allowed people with psychological disorders to be hospitalised. Notably, the people who were hospitalised were not described as being possessed (Allderidge, 1979). However, it was not until the fifteenth century that hospitals for people with psychological disorders started to appear in Europe.

Confining people with psychological disorders in hospitals and placing their care in the domain of medicine did not necessarily lead to more humane and effective treatment. Medical treatments were often crude and painful. Benjamin Rush (1745–1813), for example, began practising medicine in Philadelphia in 1769 and is considered the father of American psychiatry. Yet he believed that psychological disorder was caused by an excess of blood in the brain, for which his favoured treatment was to draw great quantities of blood from disordered individuals (Farina, 1976). Rush also believed that many people with psychological disorders could be cured by being frightened. Thus, one of his recommended procedures was for the physician to convince the patient that death was near!

In 1793, while the French Revolution raged, Philippe Pinel (1745–1826) was put in charge of a large **asylum** in Paris known as La Bicêtre. A historian described the conditions at this particular hospital.

[The patients were] shackled to the walls of their cells, by iron collars which held them flat against the wall and permitted little movement. They could not lie down at night, as a rule. Oftentimes there was a hoop of iron around the waist of the patient and in addition chains on both the hands and the feet. These chains [were] sufficiently long so that the patient could feed himself out of a bowl, the food usually being a mushy gruel — bread soaked in a weak soup. Since little was known about dietetics, [no attention] was paid to the type of diet given the patients. They were presumed to be animals and not to care whether the food was good or bad. (Selling, 1940, p. 54)

Jean-Baptiste Pussin, who was a former patient and had become an orderly at La Bicêtre during the time of Pinel, is credited with removing the chains of people imprisoned in the hospital. Pinel came to believe that people in his care were first and foremost human beings, and thus these people should be approached with compassion and understanding, and treated with dignity. He surmised that if their reason had left them because of severe personal and social problems, it might be restored to them through comforting counsel and purposeful activity. Some people who had been incarcerated for years were apparently restored to health and eventually discharged from the hospital after having been accommodated in inviting rooms and allowed to stroll freely through the hospital grounds. However, while Pinel did much good for people with psychological disorders, he reserved the more humanitarian treatment for the upper classes rather than those of the lower classes who were still subjected to terror and coercion as a means of control, with straitjackets replacing chains. This is an important issue, as stigma also appears to affect people from some social groups more than others.

Elsewhere, such as in the United States, private hospitals such as the Friends' Asylum, founded in 1817 in Pennsylvania, and the Hartford Retreat, instituted in 1824 in Connecticut, were also established to provide humane treatment. In accordance with an approach that became known as **moral treatment** — where affected people had close contact with attendants, who talked and read to them and encouraged them to engage in purposeful activity — residents led lives as close to normal as possible and in general took responsibility for themselves within the constraints of their disorders. Further, there were to be no more than 250 people in a given hospital (Whitaker, 2002). Moral treatment was largely abandoned in the latter part of the nineteenth century in an attempt by mental health crusader Dorothea Dix (1802–1887) to standardise such care from private hospitals to more accessible state-based hospitals. While Dix campaigned vigorously to improve the lives of people with psychological disorders and personally helped see that 32 state hospitals were built, the large public hospitals became overcrowded and the small staff numbers were unable to provide the individual attention that was a hallmark of moral treatment (Bockhoven, 1963). Moreover, the hospitals became administered by physicians, who were largely interested in the biological and physical aspects of illness, rather than the psychological wellbeing of people with psychological disorders.

An example of the negative impact of a purely biological focus can be seen in the outcomes of pre-frontal lobotomies, introduced in 1935 by Egas Moniz, a Portuguese psychiatrist. Moniz introduced a surgical procedure that destroys the tracts connecting the frontal lobes to other areas of the brain. His initial reports claimed high rates of success (Moniz, 1936) and for 20 years thereafter thousands of

people with psychological disorders underwent variations of this psychosurgery. The procedure was used especially for those whose behaviour was violent. Many people did indeed quiet down and could even be discharged from hospitals, but largely because their brains were damaged. Many people became dull and listless and suffered serious losses in their cognitive capacities — for example, becoming unable to carry on a coherent conversation with another person — which is not surprising given the destruction of parts of their brains that support thought and language. It was not until the 1950s that this intervention fell into disrepute.

Other negative impacts on people with mental illnesses can be attributed to the **eugenics movement**. Francis Galton (1822–1911), often considered the originator of genetic twin research with his English study of twins in the late 1800s, attributed many behavioural characteristics to heredity. He is credited with coining the terms *nature* and *nurture* to talk about differences in genetics (nature) and environment (nurture). In the early twentieth century, investigators became intrigued by the idea that psychological disorders may run in families, and beginning at that time, a number of studies documented the heritability of psychological disorders such as schizophrenia, bipolar disorder and depression. These studies would set the stage for later theories about the causes of psychological disorders. Unfortunately, Galton is also credited with creating the eugenics movement in 1883 (Brooks, 2004), which sought to eliminate undesirable characteristics from the population by restricting the ability of certain people to have children (e.g., by enforced sterilisation). Many of the early efforts to determine whether psychological disorders could be inherited were associated with the eugenics movement and this stalled research progress. State laws in the late 1800s and early 1900s in the United States prohibited people with psychological disorders from marrying and forced them to be sterilised in order to prevent them from ‘passing on’ their illness. Such laws were upheld by the US Supreme Court in 1927 (Chase, 1980), and it wasn’t until the middle of the twentieth century that these abhorrent practices were halted. Nevertheless, much damage had been done: by 1945, more than 45 000 people with psychological disorders in the United States had been forcibly sterilised (Whitaker, 2002). In Australia, as late as 1997, a formal government report showed that permission, based on medical grounds, was given by the Family Court and other state-based guardianship tribunals for only very few forced sterilisation procedures to be carried out on people with disabilities; however, other figures showed that a much greater number of sterilisations were actually carried out in the same period from 1992 to 1997 (see Brady, Britton, & Grover, 2001; Hallahan, 2012). The United Nations raised concerns about such practices, as well as concerns about Australia’s purported breach of the human rights of Indigenous people and asylum seekers (United Nations, 2015). The potential impact on inhumane management of specific populations by eugenics-based attitudes needs particular attention by ethical practitioners.

The search for biological causes dominated the field of psychopathology until well into the twentieth century, beginning in the late eighteenth century, but has since had a re-emergence with a different set of ethical frameworks, no doubt influenced by psychological and humanistic perspectives, which are covered in the following sections of this chapter. Various psychological points of view have emerged that attribute psychological disorders to psychological malfunctions. These theories were fashionable first in France and Austria and later in the United States, leading to the development of psychotherapeutic interventions based on the tenets of the individual theories.

## 1.3 Early psychological approaches to psychopathology

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**LEARNING OUTCOME 1.3** Understand early psychological approaches to psychopathology.

During the eighteenth century in Western Europe, many people were observed to be subject to hysteria, which included physical incapacities such as blindness or paralysis for which no physical cause could be found. Franz Anton Mesmer (1734–1815) believed that hysteria was caused by a particular distribution of a universal magnetic fluid in the body. Moreover, he felt that one person could

influence the fluid of another to bring about a change in the other's behaviour. Mesmer conducted meetings cloaked in mystery and mysticism, at which afflicted people sat around a covered wooden tub, with iron rods protruding through the cover from bottles underneath that contained various chemicals. Mesmer would enter the room, take various rods from the tub and touch afflicted parts of a person's body. The rods were believed to transmit animal magnetism and to adjust the distribution of the universal magnetic fluid, thereby removing the hysterical disorder. Later, Mesmer perfected his routines by simply looking at people rather than using rods. Whatever we may think of this questionable explanation and strange procedure, Mesmer apparently helped many people overcome their hysterical problems.

Although Mesmer regarded hysteria as having strictly biological causes, we discuss his work here because he is generally considered one of the earlier practitioners of modern-day **hypnosis**. (The word **mesmerise** is a synonym for hypnotise; the phenomenon itself was known to the ancients of many cultures, where it was part of the sorcery and magic of conjurers and faith healers.) Hypnosis gradually became respectable with the great Parisian neurologist Jean Martin Charcot (1825–1893) also studying hysterical states. Although Charcot believed that hysteria was a problem with the nervous system and had a biological cause, he was also persuaded by psychological explanations. One day, some of his enterprising students hypnotised a healthy woman and, by suggestion, induced her to display certain hysterical symptoms. Charcot was deceived into believing that she was an actual patient with hysteria. When the students showed him how readily they could remove the symptoms by waking the woman, Charcot became interested in psychological interpretations of these very puzzling phenomena. Given Charcot's prominence in Parisian society, his support of hypnosis as a worthy treatment for hysteria helped to legitimise this form of treatment among medical professionals of the time (Harrington, 2008; Hustvedt, 2011).

Later, in the nineteenth century, Viennese physician Josef Breuer (1842–1925) treated a young woman, whose identity was disguised under the pseudonym Anna O, with a number of hysterical symptoms, including partial paralysis, impairment of sight and hearing and, often, difficulty speaking. She also sometimes went into a dreamlike state or 'absence', during which she mumbled to herself, seemingly preoccupied with troubling thoughts. Breuer hypnotised her, and while hypnotised, she began talking more freely and, ultimately, with considerable emotion about upsetting events from her past. Frequently, on awakening from a hypnotic session she felt much better. Breuer found that the relief of a particular symptom seemed to last longer if, under hypnosis, she was able to recall the event associated with the first appearance of that symptom and if she was able to express the emotion she had felt at the time. Reliving an earlier emotional trauma and releasing emotional tension by expressing previously forgotten thoughts about the event was called **catharsis**.

In 1895, Breuer and a younger colleague, Sigmund Freud (1856–1939), jointly published *Studies in Hysteria*, partly based on the case of Anna O. The case of Anna O became one of the best-known clinical cases in the psychoanalytic literature. Ironically, later investigation revealed that Breuer and Freud reported the case incorrectly. Historical study by Ellenberger (1972) indicates that Breuer's talking cure helped the young woman only temporarily. This claim is supported by Carl Jung (1875–1961), a renowned colleague of Freud's, who is quoted as saying that during a conference in 1925, Freud told him that Anna O had never been cured. Hospital records discovered by Ellenberger confirmed that Anna O continued to rely on morphine to ease the 'hysterical' problems that Breuer is reputed to have cured by catharsis.

Nonetheless, the apparently powerful role played by factors of which people seemed unaware led Freud to postulate that much of human behaviour is determined by forces that are inaccessible to awareness. The central assumption of Freud's theorising, often referred to as **psychoanalytic theory**, is that psychopathology results from **unconscious conflicts** in the individual.

Freud divided the mind, or the **psyche**, into three principal parts: **id**, **ego** and **superego**. According to Freud, the id is present at birth and is the repository of all of the energy needed to run the psyche, including the basic urges for food, water, elimination, warmth, affection and sex. Trained as a neurologist, Freud saw the source of the id's energy as biological and he called this energy **libido**. The individual cannot consciously perceive this energy — it is **unconscious**, below the level of awareness. The id seeks immediate gratification of its urges, operating on what Freud called the **pleasure principle**. When the id is not satisfied, tension is produced and the id impels a person to eliminate this tension as quickly as possible. For example, a baby feels hunger and is impelled to move about, sucking, in an attempt to reduce the tension arising from the unsatisfied drive. A person may also attempt to obtain gratification by generating images — in essence, fantasies — of what is desired. For instance, the hungry baby imagines sucking at the mother's breast and thereby obtains some substitute, short-term satisfaction. Of course, fantasising cannot really satisfy such urges. This is where the ego comes in.

According to Freud, the ego begins to develop from the id during the second six months of life. Unlike the contents of the id, those of the ego are primarily conscious. The id may resort to fantasy when seeking satisfaction, but the task of the ego is to deal with reality. The ego thus operates on what Freud termed the **reality principle** as it mediates between the demands of reality and the demands of the id for immediate gratification. The superego — the third part of the psyche in Freud's **theory** — can be roughly conceived of as a person's **conscience**. Freud believed that the superego develops throughout childhood, arising from the ego much as the ego arises from the id. As children discover that many of their impulses — for example, biting and bed-wetting — are not acceptable to their parents, they begin to incorporate parental values as their own in order to receive the pleasure of parental approval and avoid the pain of disapproval. Freud conceived of the personality as developing through a series of four distinct psychosexual stages (see focus on discovery 1.1). He used the term psychosexual because, at each stage, a different part of the body is the most sensitive to sexual excitation and, therefore, the most capable of satisfying the id.

According to Freud, and later elaborated on by his daughter Anna Freud (1946/1966), herself an influential psychoanalyst, discomfort experienced by the ego as it attempts to resolve conflicts and satisfy the demands of the id and superego can be reduced in several ways. A **defence mechanism** is a strategy used by the ego to protect itself from anxiety. Examples of defence mechanisms are presented in table 1.1.

#### FOCUS ON DISCOVERY 1.1

##### Stages of psychosexual development

Freud conceived of the personality as developing through a series of five distinct psychosexual stages. He used the term *psychosexual* because, at each stage, a different part of the body is the most sensitive to sexual excitation and, therefore, the most capable of satisfying the id.

During the **oral stage**, which lasts from birth to about 18 months, the demands of an infant's id are satisfied primarily by feeding, and the sucking and biting associated with it. The body parts through which the infant receives gratification at this stage are the lips, mouth, gums and tongue. During the **anal stage**, from about 18 months to 3 years of age, a child receives pleasure mainly via the anus, by passing and retaining faeces. The **phallic stage** extends from age 3 to age 5 or 6; during this stage, maximum gratification of the id is obtained through genital stimulation. Between the ages of 6 and 12, the child is in a **latency period**; during these years the id impulses do not play a major role in motivating behaviour. The final and adult stage is the **genital stage**, during which heterosexual interests predominate.

During each stage, the developing person must resolve the conflicts between what the id wants and what the environment will provide. How this is accomplished is believed, in Freud's view, to determine basic personality traits that last throughout the person's life. A person who experiences either excessive or deficient amounts of gratification at a particular stage develops a **fixation** and is likely to regress to that stage when stressed.

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According to Freud, too much or too little gratification during one of the psychosexual stages may lead to regression to this stage during stress.



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In Freud's theory, the first stage of psychosexual development is the oral stage, during which pleasure is obtained from feeding.



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### QUESTIONS

If you run down a list of obscenities — taboo words — you will find that most reflect one or another of Freud's stages.

1. How might Freud have explained this?
2. What other explanations could make sense of this phenomenon?

**TABLE 1.1** Selected defence mechanisms

Defence mechanism	Definition	Example
Repression	Keeping unacceptable impulses or wishes from conscious awareness	A professor starting a lecture she dreaded giving says, 'In conclusion'.
Denial	Not accepting a painful reality into conscious awareness	A victim of childhood abuse does not acknowledge it as an adult.
Projection	Attributing to someone else one's own unacceptable thoughts or feelings	A man who hates members of a racial group believes that it is they who dislike him.
Displacement	Redirecting emotional responses from their real target to someone else	A child gets mad at her brother but instead acts angrily towards her friend.
Reaction formation	Converting an unacceptable feeling into its opposite	A person with sexual feelings towards children leads a campaign against child sexual abuse.
Regression	Retreating to the behavioural patterns of an earlier stage of development	An adolescent dealing with unacceptable feelings of social inadequacy attempts to mask those feelings by seeking oral gratification.
Rationalisation	Offering acceptable reasons for an unacceptable action or attitude	A parent berates a child out of impatience, then indicates that she did so to 'build character'.
Sublimation	Converting unacceptable aggressive or sexual impulses into socially valued behaviours	Someone who has aggressive feelings towards his father becomes a surgeon.

Psychological treatment that emanates from Freud's theory, specifically **psychoanalysis** or psychoanalytic therapy, is based on helping affected individuals resolve their unconscious conflicts and use more adaptive defences. It is practised today across many parts of the world, and has made a recent comeback on the basis of evidence that it is effective in some instances (Luyten, Mayes, Fonagy, Target, & Blatt, 2015). In psychoanalysis and the later psychodynamic treatments, the goal of the therapist is to understand the person's early-childhood experiences, the nature of key relationships, the patterns in current relationships and unhelpful defence mechanisms. The therapist is listening for core emotional and relationship themes that surface again and again (see table 1.2 for a summary of psychoanalytic techniques).

**TABLE 1.2** Major techniques of psychoanalysis

Technique	Description
Free association	A person tries to say whatever comes to mind without censoring anything.
Interpretation	The analyst points out the meaning of a person's behaviour.
Analysis of transference	The person responds to the analyst in ways that the person has previously responded to other important figures in his or her life, and the analyst helps the person understand and interpret these responses.

Several of Freud's contemporaries met with him periodically to discuss psychoanalytic theory and therapy. As often happens when a brilliant leader attracts brilliant followers and colleagues, disagreements arose about many general issues, such as the relative importance of id versus ego, of biological

versus sociocultural forces on psychological development, of unconscious versus conscious processes and of childhood versus adult experiences; whether sexual urges drive behaviours that are not obviously sexual; and the role of reflex-like id impulses versus that of purposeful behaviour governed primarily by conscious ego deliberations. Two particularly influential historical figures were Carl Jung and Alfred Adler.

Carl Gustav Jung (1875–1961), a Swiss psychiatrist originally considered Freud's heir apparent, broke with Freud in 1914 on many issues. This followed a seven-year period of intense correspondence about their disagreements. Jung proposed ideas radically different from Freud's, ultimately establishing **analytical psychology**. Jung hypothesised that in addition to the personal unconscious postulated by Freud, there is a **collective unconscious**, the part of the unconscious that is common to all human beings, and that consists primarily of what Jung called **archetypes**, or basic categories that all human beings use in conceptualising the world. In addition, Jung asserted that each of us has masculine and feminine traits that are blended and that people's spiritual and religious urges are as basic as their id urges. Jung also catalogued various personality characteristics; perhaps most important among them are **extraversion** (an orientation towards the external world) versus **introversion** (an orientation towards the inner, subjective world). This personality dimension continues to be regarded as very important.

Alfred Adler (1870–1937), also an early adherent of Freud's theories, came to be even less dependent on Freud's views than was Jung, and Freud remained quite bitter towards Adler after their relationship ended. Adler's theory, which came to be known as **individual psychology**, regarded people as inextricably tied to their society because he believed that fulfilment was found in doing things for the social good. Like Jung, he stressed the importance of working towards goals (Adler, 1930). A central element in Adler's work was his focus on helping people change their illogical and mistaken ideas and expectations. He believed that feeling and behaving adaptively depend on thinking more rationally, an approach that anticipated contemporary developments in cognitive–behavioural therapy (discussed below).

Freud's original ideas and methods have been heavily criticised over the years. Freud conducted no formal research on the causes and treatments of psychological disorders, and this remains one of the main criticisms today. Because psychoanalytic notions are based on anecdotal evidence gathered during therapy sessions, some psychodynamic theories are not grounded in objectivity and are not scientifically testable. However, other contemporary psychodynamic theories, such as object relations theory, have built a limited base of supportive research. Offshoots of object relations theory, such as attachment theory and the idea of the **self** or **identity**, have accumulated a good bit of research support, both in children and adults. All of these concepts are discussed later in this chapter, as they seem to transcend contemporary paradigms.

Though perhaps not as influential as it once was, the work of Freud and his followers continues to have an impact on the field of psychopathology (Westen, 1998) and has impacted positively on stigma by emphasising the importance of experiential and developmental factors in the aetiology of psychological disorder. This influence is most evident in the following three commonly held assumptions.

1. *Childhood experiences help shape adult personality.* Contemporary clinicians and researchers still view childhood experiences and other environmental events as crucial. They seldom focus on the psychosexual stages about which Freud wrote, but some emphasise problematic parent–child relationships in general and how they can influence later adult relationships in negative ways.
2. *There are unconscious influences on behaviour.* The unconscious is a focus of contemporary research in cognitive neuroscience and psychopathology. This research shows that people can be unaware of the causes of their behaviour. However, most current researchers and clinicians do not think of the unconscious as a repository of id instincts.
3. *The causes and purposes of human behaviour are not always obvious.* Freud and his followers sensitised generations of clinicians and researchers to the non-obviousness of the causes and purposes of human behaviour. Contemporary psychodynamic theorists continue to caution us against taking everything at face value. A person expressing disdain for another, for example, may actually like the

other person very much, yet be fearful of admitting positive feelings. This tendency to look under the surface, to find hidden meanings in behaviour, is perhaps Freud's best-known legacy.

Hence, as can be seen, theories about psychopathology and the treatment of people with psychological disorders has changed over time, though not always for the better, nor always based on scientific evidence.

Of particular concern for researchers and clinicians is the need to recognise our own preconceived notions of psychological disorders, and the need to confront and work to change the stigma we often associate with mental health conditions. As has been seen throughout recorded history, the treatment of people with psychological disorders has not generally been good or effective, and this has contributed to their stigmatisation, to the extent that they have often been brutalised and shunned by society. In the past, torturous treatments were held up to the public as miracle cures and, even today, terms such as 'crazy', 'insane', 'retard' and 'schizo' are tossed about without thought for the people who actually suffer from psychological disorders and for whom these insults and the intensely distressing feelings and behaviours they refer to are a reality of daily life.

Throughout this text, we hope to fight this stigma by showing you the latest evidence about the nature and causes of these disorders, together with treatments, dispelling myths and other misconceptions as we proceed. As part of this effort, we will try to put a human face on psychological disorders by including descriptions of actual people with these disorders.

But you will have to help in this fight, for the mere acquisition of knowledge does not ensure the end of stigma (Penn, Chamberlin, & Mueser, 2003). As we will see, in the last 20 years we have learned a great deal about social, neurobiological and experiential contributors to psychological disorders, including neurotransmitters, genetics, brain functions and stigma itself. Many mental health practitioners and advocates hoped that the more people learned about the broad causes of psychological disorders, the less stigmatised these disorders would be. However, this may not be the case. As an example, results from one study show that, even though community knowledge has increased, unfortunately stigma has not decreased (Pescosolido et al., 2010). In the study, researchers surveyed people's attitudes and knowledge about psychological disorders at two points in time: 1996 and 2006. Compared to 1996, people in 2006 were more likely to believe that psychological disorders such as schizophrenia, depression and alcohol addiction had a neurobiological cause, but stigma towards these disorders did not decrease. In fact, in some cases it increased. For example, people in 2006 were less likely to want to have a person with schizophrenia as their neighbour compared to people in 1996. We still see similar situations even in today's world. For instance, despite the plethora of shows on television about people with hoarding problems, there are still significant degrees of stigma associated with hoarding disorder (Mataix-Cols, Billotti, Fernández de la Cruz, & Nordsletten, 2013). Clearly, there is work to be done to reduce stigma, and overly focusing on one specific paradigm over the others may not be the best strategy to diminish it.

The following sections give an overview of the major paradigms used to understand psychopathology.

## 1.4 The genetic paradigm

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**LEARNING OUTCOME 1.4** Describe the essentials of the genetic paradigm.

Genes do not, on their own, make us smart, dumb, sassy, polite, depressed, joyful, musical, tone-deaf, athletic, clumsy, literary or incurious. Those characteristics come from a complex interplay within a dynamic system. Every day in every way you are helping to shape which genes become active. Your life is interacting with your genes. (Shenk, 2010, p. 27)

Over the past 20 years, researchers have decoded the human genome. That exciting milestone has been augmented with the virtual explosion of information regarding human genetics. The changes that have occurred in the **genetic paradigm** have transformed the way we think about genes and behaviour. We no longer have to wonder, 'Is nature or nurture responsible for human behaviour?' We now know that (1) almost all behaviour is heritable to some degree (i.e., it involves genes) and (2) despite this, genes do

not operate in isolation from the environment. Instead, throughout the life span, the environment shapes how our genes are expressed and our genes also shape our environments (Jaffee & Price, 2007; Kendler, 2013; Plomin, DeFries, Craig, & McGuffin, 2003; Nestler, Peña, Kundakovic, Mitchell, & Akbarian, 2016; Rutter & Silberg, 2002; Turkheimer, 2000).

The current way to think about genes and the environment is cast as ‘nature via nurture’ (Ridley, 2003). In other words, researchers are learning how environmental influences, such as **stress**, relationships and culture (the nurture part), shape which of our genes are turned on or off, and how our genes (the nature part) influence our bodies and brain. We know that without genes, a behaviour might not be possible. But without the environment, genes could not express themselves and thus contribute to the behaviour.

When the ovum, the female reproductive cell, is joined by the male’s sperm, a zygote, or fertilised egg, is produced. It has 46 **chromosomes**, the number characteristic of a human being. Each chromosome is made up of many **genes**, the carriers of the genetic information (DNA) passed from parents to child.

People have around 20 000 genes (Human Genome Project, 2008; Moraes & Goes, 2016). At first, this news was surprising; since the mere fruit fly has around 14 000 genes, researchers had thought that surely human beings were several times more complex than that! As it turns out, however, the number of genes is not all that important. Instead, it is the sequencing, or ordering, of these genes as well as what the genes actually *do* that make us unique. What genes do is make proteins that in turn make the body and brain work. Some of these proteins switch other genes on and off, a process called **gene expression**. Learning about the flexibility of genes and how they switch on or off has closed the door on beliefs about the inevitability of the effects of genes, good or bad.

With respect to most psychological disorders, there is not one gene that contributes vulnerability. Instead, psychopathology is **polygenic**, meaning several genes, perhaps operating at different times during the course of development, turning themselves on and off as they interact with a person’s environment, is the essence of genetic vulnerability. It is also likely that there are commonalities among various disorders in their genetic profiles. For instance, Bulik-Sullivan et al. (2015) concluded there were genetic correlations between anorexia nervosa and schizophrenia, anorexia and obesity, and educational attainment and several diseases. One Australian study that reviewed 134 specific genes previously identified as linked to five specific mental disorders with core emotional and cognitive dysfunction found no genetic effects for 195 genes, while 13 genetic variants were shared in common between two or more of the five disorders (Gatt, Burton, Williams, & Schofield, 2015). Although this study demonstrates evidence for multiple effects from single genes, numerous other studies of the same disorders have reported no overlap in genetic variants. Determining genetic associations between complex traits and disorders can identify potential causal gene-behaviour relationships. However, such research is in its infancy and requires replications to determine the reasons underlying inconsistent findings.

An important term that will be used throughout the text is **heritability**. Unfortunately, it is a term that is easily misunderstood and often misused. Heritability refers to the extent to which variability in a particular behaviour (or disorder) in a population can be accounted for by genetic factors. There are two important points about heritability to keep in mind:

1. heritability estimates range from 0.0 to 1.0: the higher the number, the greater the heritability
2. heritability is relevant *only* for a large population of people, not a particular individual.

Thus, it is incorrect to talk about any one person’s heritability for a particular behaviour or disorder. Knowing that the heritability of schizophrenia is estimated to range from 0.65 to 0.80 (Lichtenstein et al., 2009; Sullivan, Kendler, & Neale, 2003) does not mean that 65 to 80 percent of Emma’s schizophrenia is the product of her genes and 20 to 35 percent is the product of other factors. Rather, it means that in a population (e.g., a large sample in a study), the variation in schizophrenia is understood as being attributed to 65 to 80 percent genetic factors and 20 to 35 percent environmental factors. There is no heritability in schizophrenia (or any disorder) for a particular individual.

We now know that we do not inherit psychological disorders from our genes alone; we develop them through the interaction of our genes with our environments (Shenk, 2010). This is a subtle but very important point. It is easy to fall into the trap of thinking a person inherits a disorder like schizophrenia

from his or her genes. What contemporary genetics research today is telling us, however, is that a person *develops* schizophrenia from the interaction between genes and the environment, as well as the body (e.g., hormones, the brain and other genes).

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Shared environment refers to things families have in common, like marital quality.



Other factors that are just as important as genes in genetic research are environmental factors. **Shared environment** factors include those things that members of a family have in common, such as family income level, child-rearing practices and parents' marital status and quality. **Non-shared environment** (sometimes referred to as *unique environment*) factors are those things believed to be distinct among members of a family, such as relationships with friends or specific events unique to a person (e.g., being in a car accident or on the local cricket team) and these are believed to be important in understanding why two siblings from the same family can be so different. Consider an example. Jason is a 34-year-old man living in Auckland who is dependent on alcohol and struggling to keep his job. His sister Joan is a 32-year-old executive in a computer company in Singapore and has no alcohol or drug problems. Jason did not have many friends as a child; Joan was one of the popular girls in high school. Jason and Joan shared several influences, including their family atmosphere growing up. They also had unique, non-shared experiences, such as differences in peer relationships. Behaviour genetics research suggests that the non-shared, or unique, environmental experiences have much more to do with the development of psychological disorders than the shared experiences.

Using behavioural and survey data, the Australian Temperament Project (see Vassallo & Sanson, 2013, for a summary) has also established the importance of individual differences among siblings in temperamental characteristics, evident from infancy and considered to have a significant genetic contribution (e.g., sociability/shyness, reactivity and persistence/distractibility). These differences impact on the development of unique child–parent and child–environment transactions. Jason and Joan from the example above had very different temperaments from birth, which are likely to have differentially influenced their parents' reactions, as well as the responses of others in their broader environment. While Jason was shy, over-reactive and distractible as an infant, Joan was outgoing, flexible and persistent. These idiosyncratic

characteristics influenced how their parents, siblings, friends, peers and teachers interacted with them which, in turn, impacted differentially on, the quality and nature of their social relationships, the coping strategies that they were able to develop and, ultimately, their self-esteem. Hence, Joan and Jason were able to generate unique environments for themselves even though they grew up in the same family and sociocultural context.

We now turn to review two broad approaches in the genetic paradigm, including behaviour genetics and molecular genetics. We then discuss the exciting evidence on the ways in which genes and environments interact. This sets the stage for our discussion of an integrative paradigm later in the chapter.

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Non-shared environment refers to factors that are distinct among family members, such as having different groups of friends.



## Behaviour genetics

**Behaviour genetics** is the study of the *degree* to which genes and environmental factors influence behaviour. To be clear, behaviour genetics is not the study of *how* genes or the environment determine behaviour. Many behaviour genetics studies estimate the heritability of a psychological disorder without providing any information about how the genes might work. The total genetic make-up of an individual, consisting of inherited genes, is referred to as the **genotype** (physical sequence of DNA); the genotype cannot be observed outwardly. In contrast, the totality of observable behavioural characteristics, such as level of anxiety, is referred to as the **phenotype**.

We defined gene expression earlier: the genotype should not be viewed as a static entity. Genes switch off and on at specific times, for example, to control various aspects of development. Indeed, genetic programs are quite flexible — they respond in remarkable ways to things that happen to us.

The phenotype changes over time and is the product of an interaction between the genotype and the environment. For example, a person may be born with the capacity for high intellectual achievement, but whether he or she develops this genetically given potential depends on environmental factors such as upbringing and education. Hence, intelligence is an index of the phenotype.

A number of studies have demonstrated high heritability for IQ (e.g., Plomin, 1999). However, Turkheimer and colleagues (2003) found that heritability depends on environment. Their study included 319 twin pairs of 7-year-olds (114 identical, 205 fraternal) with many living in families either below the poverty line or with a low family income. Among the families of lower socioeconomic status (SES), 60 percent of the variability in children's IQ was attributable to the environment. Among the higher SES families, the opposite was found. That is, variability in IQ was more attributable to genes than to environment. Thus, being in an impoverished environment may have deleterious effects on IQ, whereas being in a more affluent environment may not help out all that much. It is important to point out that these interesting findings deal with IQ scores, a measure of what psychologists consider to be 'intelligence' rather than 'achievement' (we discuss this issue in more detail in the chapter on diagnosis and assessment).

Most recent evidence from twin research across a broad range of human traits confirms the substantial effects of genetic and non-shared environmental factors and the small to insignificant contributions of the shared environment (Del Giudice, 2016; Plomin, DeFries, Knopik, & Neiderhiser, 2013; Polderman et al., 2015; Turkheimer, Pettersson, & Horn, 2014). Reviewing virtually all published twin studies, Polderman et al. (2015) concluded there is no extensive contribution from the shared environment or non-additive genetic variation for around 70 percent of traits. Using sophisticated statistical simulation methods, Del Giudice (2016) further examined differential susceptibility models, which postulate that genetic variation interacts with exposure to early environmental stressors (e.g., prenatal stress hormones, maternal depression, family conflict) to generate vulnerability. His findings supported that 'sizable interaction effects involving individual differences in plasticity are plausible, but only if direct environmental effects are correspondingly weak; and that a major role of shared environmental factors is plausible in early development, but not in later development' (Del Giudice, 2016, p. 1330). Such results indicate the importance of considering the differential effects of genes and environment during specific developmental periods. There may also be important gender-related and other contributions. A following section in this chapter will discuss gene-environment interactions in greater detail.

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Behaviour genetics studies the degree to which characteristics, such as physical resemblance or psychopathology, are shared by family members with shared genes.

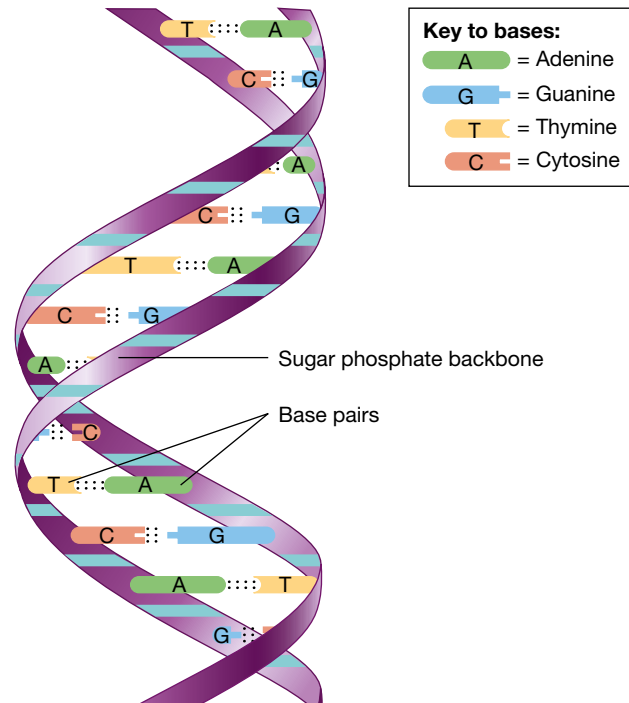


## Molecular genetics

**Molecular genetics** studies seek to identify particular genes and their functions. Recall that a human being has 46 chromosomes (23 chromosome pairs) and that each chromosome is made up of hundreds or thousands of genes that contain DNA (see figure 1.2). Different forms of the same gene are called **alleles**. The alleles of a gene are found at the same location, or locus, of a chromosome pair. A genetic **polymorphism** refers to a difference in DNA sequence on a gene that has occurred in a population.

**FIGURE 1.2**

This figure shows a strand of DNA with four chemical bases: A (adenine), T (thymine), G (guanine) and C (cytosine).

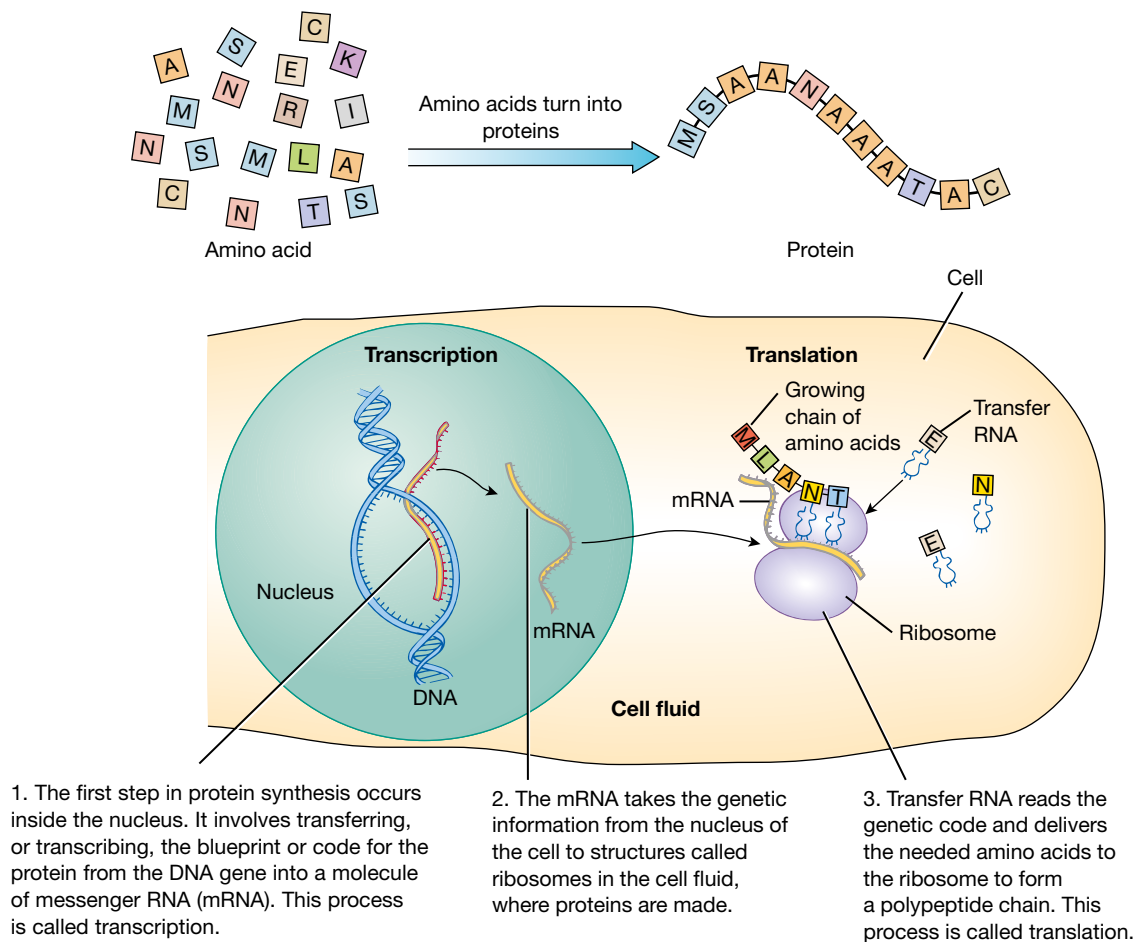


The DNA in genes is transcribed to RNA. In some cases, the RNA is then translated into amino acids, which then form proteins and proteins make cells (see figure 1.3). Gene expression involves particular types of DNA called *promoters*, recognised by particular proteins called *transcription factors*. Promoters and transcription factors are the focus of much research in molecular genetics and psychopathology. All of this is a remarkably complex system and variations along the way, such as different combinations or sequences of events, lead to different outcomes.

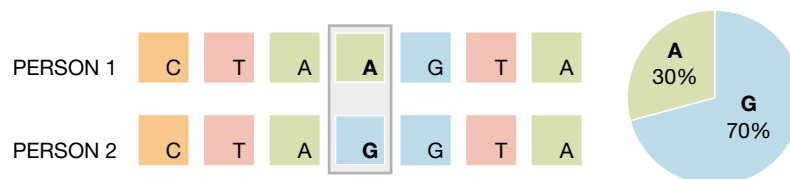
In the past 10 years, molecular genetics research has focused on identifying differences between people in the *sequence* of their genes and in the *structure* of their genes. One area of interest in the study of gene sequence involves identifying what are called **single nucleotide polymorphisms** or **SNPs** (pronounced *snips*). A SNP refers to differences between people in a single nucleotide (A, T, G or C; see figure 1.4) in the DNA sequence of a particular gene. Figure 1.2 illustrates what a SNP looks like in a strand of DNA. The SNP is circled, pointing to the single nucleotide difference between the two strands. These are the most common types of polymorphisms in the human genome, with nearly 10 million different SNPs identified so far, with each individual also holding many rare mutations. SNPs can act as biological indicators or markers, helping detect genes linked to specific disorders and tracking how heritable disorder-specific genes are within families. SNPs have been studied in schizophrenia, autism and the **mood disorders**, to name a few disorders. While most SNPs have little or no effect on health or development, there is hope that knowledge of specific genetic differences will help predict individual

response to particular medications, vulnerability to environmental stress and the risk of developing particular disorders. Evidence has also emerged from **genome-wide association studies (GWAS)**, which consider a genome-wide set of genetic variations in particular cohorts to identify whether specific variants are associated with a trait or characteristic. GWASs contrast the SNPs of individuals with different phenotypes for a specific trait. GWASs demonstrate that multiple genetic variants impact on the variation in complex traits and that effect sizes are usually small (Polderman et al., 2015). Figure 1.3 shows gene **transcription**, the process by which DNA is transcribed to RNA. In some cases, the RNA is then translated into amino acids, which then form proteins and proteins make cells.

**FIGURE 1.3** The process of gene transcription



**FIGURE 1.4** Illustration of what a single nucleotide polymorphism, or SNP, looks like in a comparison of two people. The two strands are the same except for a single nucleotide. To the right, an illustration showing that the position of G in the sequence is far more common in a large population of people than the position of A in the sequence.



Another area of interest is the study of differences between people in gene structure, including the identification of so-called **copy number variations (CNVs)**. A CNV can be present in a single gene or multiple genes. The name refers to an abnormal copy of one or more sections of DNA within the gene(s). These abnormal copies can be *additions*, where extra copies are abnormally present, or *deletions*, where copies are missing. As much as 5 percent of the human genome contains CNVs, which can be inherited from parents or can be what are called spontaneous (*de novo*) mutations — appearing for the first time in an individual. Later we will discuss studies that have identified CNVs in different disorders, particularly schizophrenia, autism and **attention-deficit hyperactivity disorder (ADHD)**.

Researchers using **animal models** (i.e., by studying animals) can actually manipulate specific genes and then observe the effects on behaviour. Specific genes can be taken out of mice DNA — these are called knockout studies because a particular gene is knocked out of the animal's system. For example, the gene that is responsible for a specific **receptor** for the neurotransmitter serotonin, called 5-HT<sub>1A</sub>, has been knocked out in mice before their birth. As adults, they show what could be described as an anxious phenotype. Interestingly, one study that employed a novel technique to knock out this gene only temporarily found that its restoration early in development prevented the development of anxious behaviour in the adult mice (Gross et al., 2002). This is a major area of molecular genetic work. Such findings can then inform human studies, although links to humans can often remain a challenge.

## Gene–environment interactions

As we noted earlier, we know now that genes and environments work together. Life experience shapes how our genes are expressed and our genes guide us in behaviours that lead to the selection of different experiences. A **gene–environment interaction** means that a given person's sensitivity to an environmental event is influenced by genes.

Take a simple (and made-up) example. If a person has gene XYZ, he or she might respond to a snakebite by developing a fear of snakes. A person without the XYZ gene would not develop a fear of snakes after being bitten. This simple relationship involves both genes (the XYZ gene) and an environmental event (snakebite).

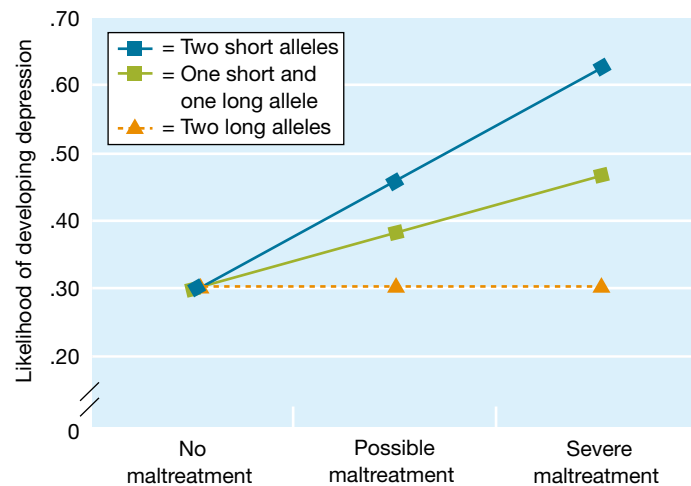
A real-life example of a gene–environment interaction involves depression. In a longitudinal study, a large sample of children in New Zealand was followed across time from the age of 5 until their mid-20s (Caspi et al., 2003). Across this time, the researchers assessed a number of variables, including early childhood maltreatment (abuse) and depression as an adult. They also measured a particular gene called the **serotonin transporter gene (5-HTT)**. This gene has a polymorphism such that some people have two short alleles (short–short), some have two long alleles (long–long) and some have one short and one long allele (short–long). Caspi and colleagues found that those individuals who had either the short–short allele or the short–long allele combinations of the 5-HTT gene *and* were maltreated as children were more likely to have **major depressive disorder (MDD)** as adults than either those people who had the same gene combination but no childhood maltreatment or those people who were maltreated as children but had the long–long allele combination of the gene (see figure 1.5). Thus, having the gene was not enough to predict an episode of depression, nor was the presence of childhood maltreatment. Rather, it was the specific combination of the gene configuration and environmental events that predicted depression. They found the same gene–environment interaction for having at least one short allele of the gene and reports of stressful life events. That is, those people who reported more severe stressful life events and had at least one short allele of the 5-HTT gene were at greater risk of developing depression.

Other recent research examining gene–environment interactions has investigated ways in which genes may impact on psychological vulnerabilities associated with mental health disorders and their treatment, as well as examining whether genetic factors mediate response to specific treatments. As a way of providing some background, in recent years, **mindfulness-based cognitive therapy (MBCT)**, based on a meditational approach to treatment, has been used to treat depression and, in particular, to prevent relapse in individuals with a history of multiple depression relapses (Segal, Williams, & Teasdale, 2013).

MBCT has been shown to increase resilience to depression. Using a large sample of adolescent twins, Waszczuk and colleagues (2015) investigated processes underlying the association between trait mindfulness, depression and anxiety sensitivity, factors considered to be significant vulnerabilities to anxiety and depression. The researchers found that around one-third of mindfulness is heritable and two-thirds influenced by non-shared environmental factors, with no impact from shared environment. They further demonstrated that common genetic factors explained more than half of the moderate magnitude relationships between the three factors of interest. Mindfulness was also distinguished by having unique genetic and environmental influences, with around two-thirds of the genetic and almost all non-shared environmental effects on mindfulness independent of depression and anxiety sensitivity. They concluded that both genes and environment assume vital roles in the emergence of mindfulness and that shared genetic vulnerability underpins the concurrent presence of low mindfulness, high-anxiety sensitivity and depression in individuals.

**FIGURE 1.5**

A gene–environment interaction is illustrated here. Having both the short allele of the 5-HTT gene and childhood maltreatment was associated with the greatest probability of developing depression as an adult.



*Source:* Adapted from Caspi et al. (2003).

Bakker et al. (2014), on the other hand, examined whether response to MBCT was mediated by SNPs associated with reward functioning. They indeed found that genetic variation in specific genes impacted on response to MBCT with change in positive affect greater in those participants with certain variants of specific SNPs, but also a decline in positive affect linked to particular SNPs for those in the control group. The authors concluded that MBCT may neutralise a genetic tendency towards deterioration in depressed individuals. Interestingly, Wichers et al. (2007) had previously concluded that, while a purported genetic vulnerability to depression is partially expressed as intense negative emotional responses to stress, this process can be moderated by individuals concurrently experiencing higher levels of positive affect after stressful events. MBCT is one way of directly facilitating such increases in positive affect. This finding highlights that processes determining outcomes are a complex amalgam rather than an inevitability linked to genetic make-up.

Animal studies also provide information about the interaction between genetic predispositions and environmental factors in the development of psychiatric disorders. In particular, numerous models of schizophrenia focusing on brain development and functioning have been developed from the ‘two hit’ hypothesis and related animal studies. Typically, rats that are genetically ‘vulnerable’ to behaviours considered analogous to human mental illnesses are subjected to environmental disruptions (e.g., prenatal viruses or stress hormones, early environmental adversity, maternal deprivation) at critical developmental

periods. Assessments are made of ‘cognitive’ abilities or brain-related chemicals in animals and their association with the behavioural dispositions is examined. The prenatal genetic or environmental ‘first hit’ is thought to disrupt some aspect of early brain development, as evidenced by cognitive deficits or brain chemistry, and generate long-term vulnerability to a ‘second hit’ that then leads to the onset of schizophrenia later in life. Neither ‘hit’ on its own is considered sufficient to induce schizophrenia.

Human research based on the ‘two hit’ hypothesis for schizophrenia has examined the interaction of genetic vulnerability with a range of vulnerabilities including malnutrition and exposure to reduced prenatal vitamin D, viral infections, social stressors and exclusion, compromised intelligence and social cognition, cannabis use, smoking and childhood trauma. A recent review of this research concluded that the ‘development of schizophrenia is likely to be more complex and nuanced than the simplistic two hit model’ (Davis et al., 2016, p. 185). In other words, the development of schizophrenia is likely to be impacted by a complex and cumulative interaction between genetic risk and numerous ‘hits’ that occur at critical periods of neurodevelopment. Impact on neurodevelopment can include alterations in specific aspects of nervous system development (e.g., creation, refinement and excessive elimination of synapses), as well as loss of **neuroplasticity**, a concept we discuss below.

Other exciting research investigates changes in behaviour and gene expression under different environmental conditions. The study of how the environment can alter gene expression or function is called **epigenetics**. The term *epigenetic* means ‘above or outside the gene’ and refers to the chemical ‘marks’ that are attached to and protect the DNA in each gene. These epigenetic marks are what control gene expression and the environment can directly influence the work of these marks (Karg, Burmeister, Shedden, & Sen, 2011; Zhang & Meaney, 2010). In a review of epigenetic influences in mental illness, Nestler and colleagues concluded that the high rates of genetic discordance among identical twins for depression, schizophrenia and bipolar disorder implicate the influence of early environmental factors, particularly stress, which prompt lasting changes in gene expression, specific brain regions and behaviour. Animal and human studies are slowly emerging that support such conclusions but we have a way to go before such findings can translate into preventative and treatment-related applications.

## Evaluating the genetic paradigm

Genetics is an important part of the study of psychopathology and there are many ways in which genes might be involved in psychopathology. Recent research has taken us a long way away from the eugenics movement with its stigmatising framework of genetic influences on psychopathology, and the models that have emerged from contemporary research will help us understand that genes do their work through the environment. There are two huge challenges facing today’s scientists working within the genetic paradigm. The first is to specify exactly how genes and environments reciprocally influence one another and which specific combinations are important to particular disorders. Currently, this is more easily done in tightly controlled laboratory studies with animals. Making the leap to humans is a greater challenge. The second is to recognise the complexity of the task, knowing that several genes (not just one) will contribute to a specific disorder and that currently there is a long pathway between the genes and the complex behaviours that make up psychological disorders, with several biological processes unfolding along the way. Putting all the small genetic pieces together in order to tell the gene via environment story for psychological disorders remains a very big challenge (Chabris et al., 2013).

Finally, as our field tends to prioritise the publication of new findings rather than replications, even if the new findings come from a study with a smaller sample size, we need to replicate findings with different populations in order to determine the validity and generalisability of results. By favouring the publication of only positive results, many studies that yield negative results do not get published, thus inflating the field’s confidence in a set of findings. This tendency to publish only positive results is called **publication bias** and as we will see in the remaining chapters, researchers are now figuring publication bias into their review of research findings. A current controversy in the field concerns how to consider these issues when interpreting the evidence for and against gene–environment interactions.

Some researchers argue that direct replications of original studies with large sample sizes should be weighted more heavily when reviewing the evidence (Duncan & Keller, 2011), whereas others contend that including indirect replication studies is important for the broader generalisability of the findings (Caspi, Hariri, Holmes, Uher, & Moffitt, 2010; Karg et al., 2011).

This is an exciting time for genetics research, and important discoveries about genes, environments and psychopathology are being made at a rapid rate. In addition, some of the most exciting breakthroughs in genetics have involved a combination of methods from genetics and neuroscience. Although we present the genetic and neuroscience paradigms separately, they go hand in hand when it comes to understanding the possible causes of psychopathology.

## 1.5 The neuroscience paradigm

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**LEARNING OUTCOME 1.5** Describe the essentials of the neuroscience paradigm.

Cognitive neuroscience is entering an exciting era in which new technologies and ideas are making it possible to study the neural basis of cognition, perception, memory and emotion at the level of networks of interacting neurons, the level at which we believe many of the important operations of the brain take place. (John O’Keefe, 2014, co-winner 2014 Nobel Prize for Medicine)

Enormous strides have been made in understanding the chemical and electrical processes that nerve cells use to communicate with each other, but much remains to be discovered. One of the greatest mysteries is the process by which this extremely complicated network generates conscious awareness, perception, behaviour and emotions. And how it does this at the same time as processing sensory input and controlling muscles and all other organs. (Fred Mendelsohn, 2013, Florey Neuroscience Institute, University of Melbourne)

In the previous section, we summarised the genetic paradigm, which, as we have seen, is often combined with the neuroscience paradigm. The **neuroscience paradigm** specifically holds that psychological disorders are linked to aberrant processes in the brain and nervous system. Considerable literature deals with the importance of specific aspects of the brain in psychopathology. For example, features of depression are associated with neurotransmitter problems; anxiety disorders may be related to a problem within the autonomic nervous system; dementia can be traced to impairments in structures of the brain or neurons. In this section, we look at three components of this paradigm in which the data are particularly interesting: neurons and neurotransmitters, brain structure and function, and the neuroendocrine system. We then consider some of the key treatments that follow from the paradigm.

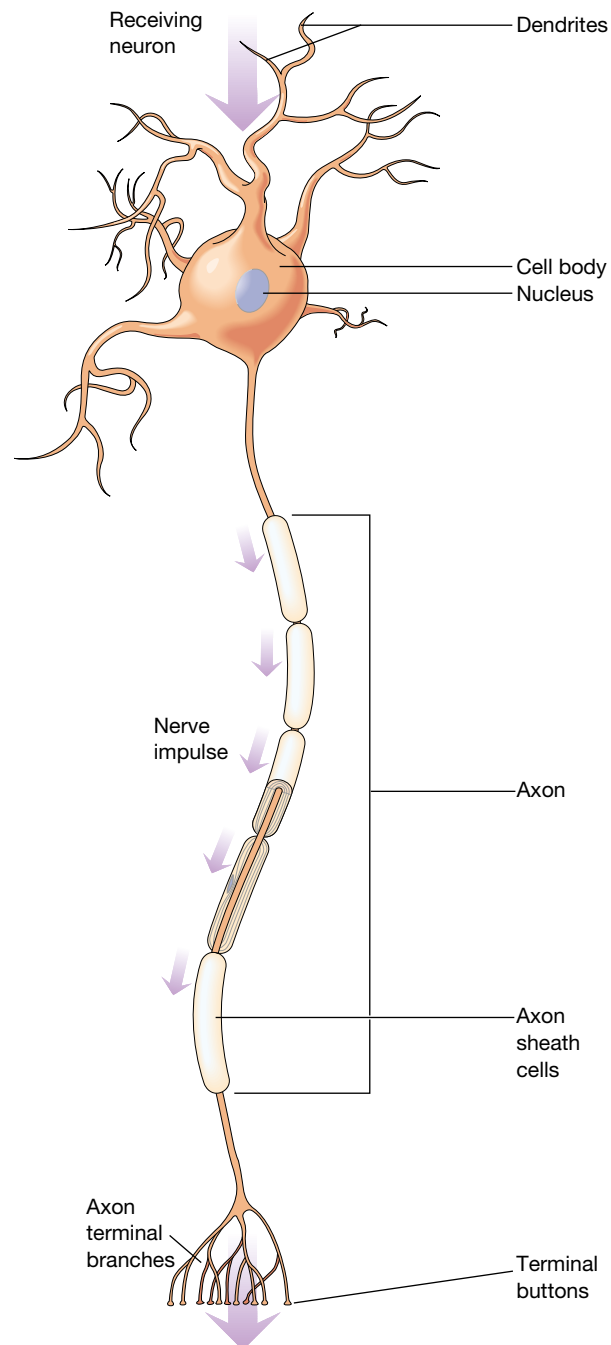
### Neurons and neurotransmitters

The cells in the nervous system are called neurons and the nervous system is composed of billions of neurons. Although neurons differ in some respects, each **neuron** has four major parts: (1) the cell body; (2) several dendrites, the short and thick extensions; (3) one or more axons of varying lengths, but usually only one long and thin axon that extends a considerable distance from the cell body; and (4) terminal buttons on the many end branches of the axon (see figure 1.6). When a neuron is appropriately stimulated at its cell body or through its dendrites, a nerve impulse travels down the axon to the terminal endings. Between the terminal endings of the sending axon and the cell membrane of the receiving neuron there is a small gap, called the **synapse** (see figure 1.7).

For neurons to send a signal to the next neuron so that communication can occur, the nerve impulse must have a way of bridging the synaptic space. The terminal buttons of each axon contain synaptic vesicles, small structures that are filled with **neurotransmitters**. Neurotransmitters are chemicals that allow neurons to send a signal across the synapse to another neuron. As the neurotransmitter flows into the synapse, some of the molecules reach the receiving, or postsynaptic, neuron. The cell membrane of the postsynaptic neuron contains receptors. Receptors are configured so that only specific neurotransmitters

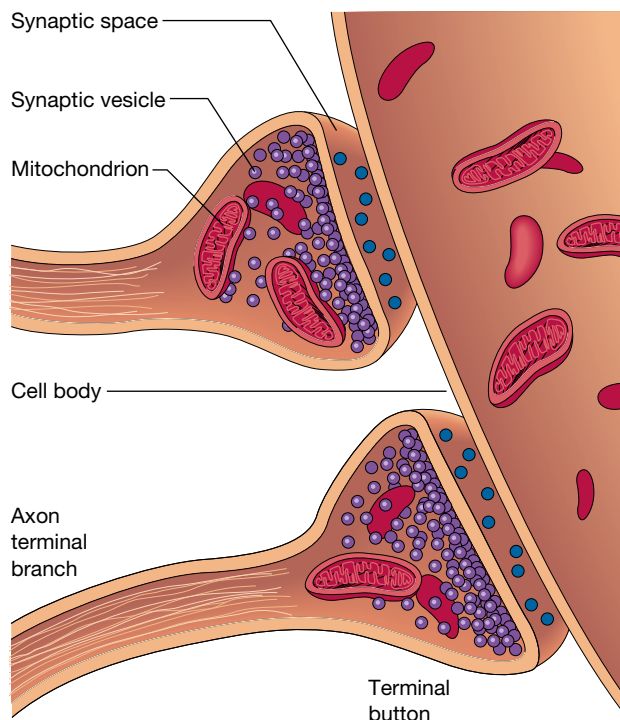
can fit into them. When a neurotransmitter fits into a receptor site, a message can be sent to the postsynaptic cell. What actually happens to the postsynaptic neuron depends on integrating thousands of similar messages. Sometimes these messages are excitatory, leading to the creation of a nerve impulse in the postsynaptic cell; at other times the messages are inhibitory, making the postsynaptic cell less likely to create a nerve impulse.

**FIGURE 1.6** The neuron, the basic unit of the nervous system



**FIGURE 1.7**

A synapse, showing the terminal buttons of two axon branches in close contact with a very small portion of the cell body of another neuron



Once a presynaptic neuron (the sending neuron) has released its neurotransmitter, the last step is for the synapse to return to its normal state. Not all of the released neurotransmitter has found its way to postsynaptic receptors. Some of what remains in the synapse is broken down by **enzymes** and some is taken back into the presynaptic cell through a process called **reuptake**.

Several key neurotransmitters have been implicated in psychopathology, including **dopamine, serotonin, norepinephrine** and **gamma-aminobutyric acid (GABA)**. Serotonin and dopamine may be involved in depression, mania and schizophrenia. Norepinephrine is a neurotransmitter that communicates with the sympathetic nervous system, where it is involved in producing states of high arousal and thus may be involved in the anxiety disorders and other stress-related conditions (see focus on discovery 1.2 for more on the sympathetic nervous system). GABA inhibits nerve impulses throughout most areas of the brain and may be involved in the anxiety disorders.

Early theories linking neurotransmitters to psychopathology sometimes proposed that a given disorder was caused by either too much or too little of a particular transmitter (e.g., mania is associated with too much norepinephrine, anxiety disorders with too little GABA). Later research has uncovered the details behind these overly simple ideas. Neurotransmitters are synthesised in the neuron through a series of metabolic steps, beginning with an amino acid. Each reaction along the way to producing an actual neurotransmitter is the outcome of catalysis by an enzyme. Too much or too little of a particular neurotransmitter could result from an error in these metabolic steps. Similar disturbances in the amounts of specific transmitters could result from alterations in the usual processes by which transmitters are deactivated after being released into the synapse. For example, a failure to pump leftover neurotransmitter back into the presynaptic cell (reuptake) would leave excess neurotransmitter in the synapse. Then, if a new nerve impulse causes more neurotransmitter to be released into the synapse, the postsynaptic neuron would, in a sense, get a double dose of neurotransmitter, making it more likely for a new nerve impulse to be created.

Other research has focused on the possibility that the neurotransmitter receptors are at fault in some disorders. If the receptors on the postsynaptic neuron were too numerous or too easily excited, the result would

be akin to having too much transmitter released. There would simply be more sites available with which the neurotransmitter could interact, increasing the chances that the postsynaptic neuron would be stimulated.

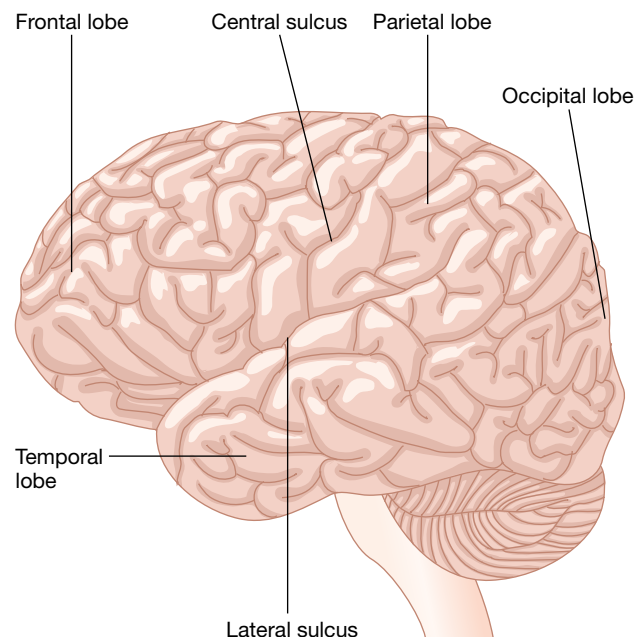
One method that investigators use to study how neurotransmitters are working in the brain is to have people take a drug that stimulates a particular neurotransmitter's receptors. This kind of drug is referred to as an **agonist**. A serotonin agonist, for example, is a drug that stimulates serotonin receptors to produce the same effects as serotonin does naturally. By contrast, an **antagonist** is a drug that works on a neurotransmitter's receptors to dampen the activity of that neurotransmitter. For example, many drugs used to treat schizophrenia are dopamine antagonists that work by blocking dopamine receptors (see the chapter on schizophrenia).

## Structure and function of the human brain

The brain is located within the protective coating of the skull and is enveloped with three protective layers of membranes referred to as meninges. The brain comprises three main parts: the forebrain, midbrain and hindbrain. The forebrain consists of the **cerebrum**, **thalamus** and **hypothalamus** and is the largest part of the brain with wide-ranging functions. The midbrain contains the tectum and tegmentum, controlling our hearing, vision, arousal, temperature regulation and motor functions. Finally, the hindbrain comprises the **cerebellum**, pons and medulla, and controls essential functions such as breathing and blood flow, which are outside our conscious control.

Viewed from the top, the brain is divided into two mirror-image cerebral hemispheres by a midline fissure; together they constitute most of the cerebrum. The major connection between the two hemispheres is a band of nerve fibres, called the **corpus callosum**, which allows the two hemispheres to communicate. Figure 1.8 shows the surface of one of the cerebral hemispheres. The outer layer of the cerebrum is called the **cerebral cortex** and comprises the neurons that form the thin outer covering of the brain, the so-called **grey matter** of the brain. The cortex consists of six layers of tightly packed neurons, estimated to number close to 16 billion. The cortex looks vastly creased with multiple folds; the ridges are called **gyri** and the indents between them **sulci** or fissures. If unfolded, the cortex would be about the size of a dinner napkin. The sulci are used to define different regions of the brain, much like guide points on a map.

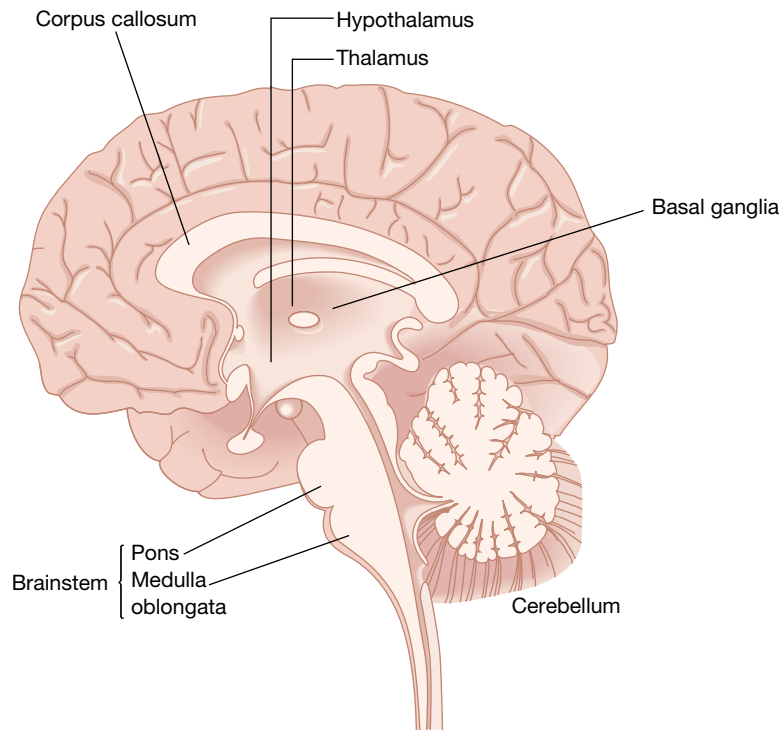
**FIGURE 1.8** Surface of the left cerebral hemisphere, showing the four lobes and the central and lateral sulci



Deep fissures divide the cerebral hemispheres into four distinct areas called *lobes*. The **frontal lobe** lies in front of the central sulcus; the **parietal lobe** is behind it and above the lateral sulcus; the **temporal lobe** is located below the lateral sulcus; and the **occipital lobe** lies behind the parietal and temporal lobes (see figure 1.8). Different functions tend to be associated with particular brain areas: vision with the occipital lobe; discrimination of sounds with the temporal lobe; reasoning, problem solving, working memory and emotion regulation with the frontal lobe. One important area of the cortex is called the **prefrontal cortex**. This region, in the very front of the cortex, helps to regulate the amygdala (discussed below) and is important in many different disorders.

If the brain is sliced in half, separating the two cerebral hemispheres, additional important structures can be seen. The grey matter of the cerebral cortex does not extend throughout the interior of the brain (see figure 1.9). Much of the interior is **white matter**, made up of large tracts of myelinated (sheathed) fibres that connect cell bodies in the cortex with those in the spinal cord and in other centres lower in the brain. In certain areas, called *nuclei*, sets of nerves converge and messages are integrated from different centres.

**FIGURE 1.9** Slice of brain showing some of the internal structures



Deep within the brain are cavities called **ventricles**. These ventricles are filled with cerebrospinal fluid. Cerebrospinal fluid circulates through the brain through these ventricles, which are connected with the spinal cord.

One important set of areas, collectively referred to as the **basal ganglia**, is located deep within each hemisphere and includes substructures such as the **caudate nucleus**, putamen, substantia nigra, subthalamic nucleus, nucleus accumbens and globus pallidus. The basal ganglia help regulate starting and stopping both motor and cognitive activity. They also regulate balance, eye movements and posture. As such, they are connected to other motor areas in the brain.

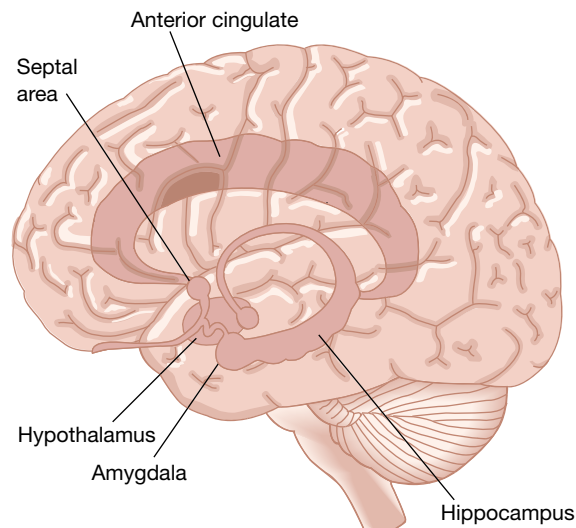
The thalamus is a structure in the middle of the brain located between the cerebral cortex and the midbrain, which acts as a relay station for all sensory pathways except the olfactory. The nuclei making

up the thalamus receive nearly all impulses arriving from the different sensory areas of the body before passing them on to the cortex, where they are interpreted as conscious sensations.

The **brainstem**, comprising the *pons* and the *medulla oblongata*, functions primarily as a neural relay station. The pons contains tracts that connect the cerebellum with the spinal cord and with motor areas of the cerebrum. The medulla oblongata serves as the main line of traffic for tracts ascending from the spinal cord and descending from the higher centres of the brain. The cerebellum receives sensory nerves from the vestibular apparatus of the ear and from muscles, tendons and joints. The information received and integrated relates to balance, posture, equilibrium and the smooth coordination of the body when in motion.

A set of deeper, mostly subcortical, structures are often implicated in different forms of psychopathology. There is a long history of referring to different groupings of these structures as the **limbic system**. These structures, shown in figure 1.10, support the visceral and physical expressions of emotion — quickened heartbeat and respiration, trembling, sweating and alterations in facial expressions — and the expression of appetitive and other primary drives, namely, hunger, thirst, mating, defence, attack and flight. Important structures are the **anterior cingulate**; the **septal area**; the **hippocampus**, which is associated with memory; the hypothalamus, which regulates metabolism, temperature, perspiration, blood pressure, sleeping and appetite; and the **amygdala**, which is an important area for attention to emotionally salient stimuli. This is one of the key brain structures for psychopathology researchers, given the ubiquity of emotional problems in the psychological disorders.

**FIGURE 1.10** Subcortical structures of the brain



The development of the human brain is a complex process that begins early in the first trimester of pregnancy and continues into early adulthood. It has been estimated that about a third of our genes are expressed in the brain and many of these genes are responsible for laying out the structure of the brain. The development of the cells and the migration of these cells to the appropriate layers of cortex comprise an intricate dance. Unfortunately, missteps can happen and current thinking about a number of disorders, such as schizophrenia, places the beginnings of the problem in these early developmental stages. Brain development continues throughout childhood, adolescence and even into adulthood. What is happening during this time is cell development and a honing of the connections between cells and brain areas. The grey matter of the brain continues to develop, filling with cells, until early adolescence. Then, somewhat surprisingly, a number of synaptic connections begin to be eliminated — a process called **pruning**. Throughout early adulthood, the connections in the brain may become fewer, but they

also become faster. The areas that develop the quickest are areas linked to sensory processes, like the cerebellum and occipital lobe. The area that develops last is the frontal lobe.

We will discuss a number of these brain areas throughout the text. For example, people with schizophrenia have been found to have enlarged ventricles of the brain (refer to the chapter on schizophrenia); the size of the hippocampus is reduced among some people with **post-traumatic stress disorder (PTSD)**, depression and schizophrenia, perhaps due to overactivity of their stress response systems (refer to the chapters of mood disorders, anxiety, obsessive-compulsive and trauma-related disorders, and schizophrenia); brain size among some children with autism expands at a much greater rate than it should in typical development (refer to the chapter on disorders of childhood).

As an example of our understanding of the association between brain regions and psychopathology, an Australian study by Yücel et al. (2007) found that, despite a lack of behavioural differences on an inhibitory control task designed to probe the medial frontal cortex, patients with **obsessive-compulsive disorder (OCD)** as compared to healthy controls showed greater activation of the supplementary motor area and deactivation of the rostral anterior cingulate during high- versus low-conflict conditions. Furthermore, OCD patients had reduced levels of neuronal N-acetylaspartate (a marker of neuronal integrity and function) in the dorsal anterior cingulate region, which was negatively correlated with their blood oxygen level-dependent activation of the region. Hyperactivation of the medial frontal cortex during the task by OCD patients may compensate for a neuronal abnormality in the region.

Finally, we need to mention the concept of neuroplasticity, reflecting the ability of the human brain to constantly change as a function of experience, a very popular notion in recent times. While the brain was once considered to be a fixed organ, it is now thought to be able to ‘re-wire’ itself. Of course, this should not come as a surprise as, without this aptitude, we would not be able to develop our abilities from infancy, childhood and adolescence through to adulthood, or to rehabilitate following injury or illness. Research shows that brain plasticity and behaviour can be influenced by multiple factors, including genes, age and experiential and environmental factors but also by natural limitations. Neuroplasticity does not equate to infinite flexibility or ongoing generation of synapses and, by adulthood, the phenomenon of synaptic pruning halves the number of synapses. Nonetheless, the architecture of the brain, while not allowing parts of the brain to take on functions that were previously unrelated to it, will still countenance rehabilitation programs reversing the damage caused, for example, in stroke by strengthening the contributions of intact connections. The mechanisms supporting neuroplasticity are still being determined and future advances will improve understanding of how we can improve outcomes following trauma.

## The neuroendocrine system

The neuroendocrine system has been implicated in psychopathology as well. One of the systems we will return to again and again is the hypothalamic-pituitary-adrenal (HPA) axis (shown in figure 1.11). The **HPA axis** is central to the body’s response to stress and stress figures prominently in many of the disorders we discuss in this text.

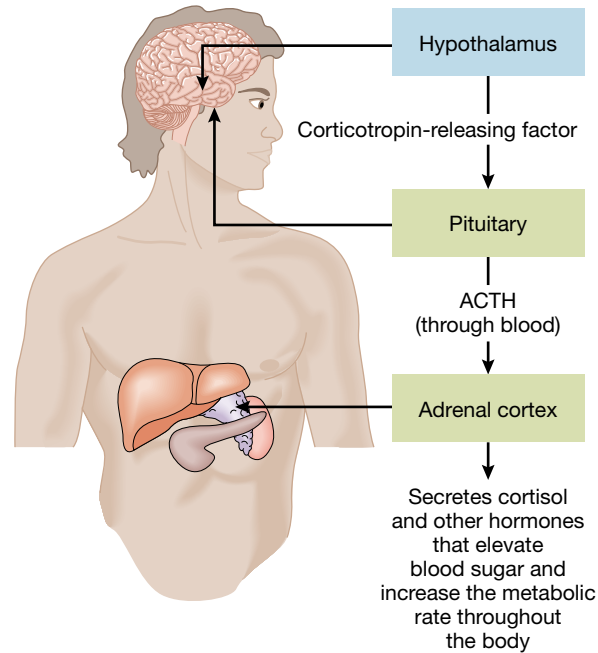
When people are faced with threat, the hypothalamus releases corticotropin-releasing factor (CRF), which then communicates with the *pituitary gland*. The pituitary then releases adrenocorticotrophic hormone, which travels via the blood to the adrenal glands. The outer layers of the adrenal glands are referred to as the *adrenal cortex*; this area promotes the release of the hormone cortisol. **Cortisol** is often referred to as the stress hormone. This is not a fast-moving system, like the autonomic nervous system, to be reviewed shortly. Rather, it takes about 20 to 40 minutes for cortisol release to peak. After the stress or threat has remitted, it can take up to an hour for cortisol to return to baseline (i.e., before the stress) levels (Dickerson & Kemeny, 2004).

Studies of stress and the HPA axis are uniquely integrative. That is, they begin with a psychological concept (stress) and examine how stress is manifested in the body (the HPA axis). For example, in a series of animal studies, researchers have shown that rats and primates that are exposed to early trauma, such as being separated from their mothers, show elevated activity in the HPA axis when they are exposed to stressors later in life (Gutman & Nemeroff, 2003). As in our discussion of gene–environment interactions earlier, it is hard to consider biology and environment separately — biology may create

increased reactivity to the environment and early experiences may influence biology. As we will see, chronic stress and its effects on the HPA axis are linked to disorders as diverse as schizophrenia, depression and post-traumatic stress disorder.

Another important system, the **autonomic nervous system (ANS)**, is discussed in focus on discovery 1.2. Much of our behaviour is dependent on a nervous system that operates very quickly, generally without our awareness and that has traditionally been viewed as beyond voluntary control; hence, the term *autonomic*.

**FIGURE 1.11** The HPA axis



## The immune system

Stress also has effects on the immune system. The field that studies how psychological factors impact the immune system is called **psychoneuroimmunology**. Research reviews have confirmed that a wide range of stressors produce problematic changes in the immune system, including medical school examinations, depression and bereavement, marital discord and divorce, job loss, caring for a relative with Alzheimer's disease and the Three Mile Island nuclear disaster, among others (Kiecolt-Glaser & Glaser, 2002; Segerstrom & Miller, 2004). As we shall see throughout, changes in the immune system are also associated with different psychological disorders.

The immune system involves a broad array of cells and proteins that respond when the body is infected or invaded. A useful way of thinking about the immune system is to consider two broad types of immunity, natural and specific (Segerstrom & Miller, 2004).

*Natural immunity* is the body's first and quickest line of defence against infectious micro-organisms or other invaders. A number of different cells, such as macrophages and natural killer cells, are unleashed on the invaders and begin to destroy them. Inflammation or swelling is a sign of these natural immunity cells at work. Activation of macrophages in turn stimulates the release of substances called **cytokines**, which help initiate such bodily responses to infection as fatigue, fever and activation of the HPA axis. Although unpleasant, fever is actually a sign that the body is responding as it should to an infection.

*Specific immunity* involves cells that respond more slowly to infection, such as lymphocytes, which are involved in responding to specific pathogens or invading agents. Lymphocytes include T-helper cells

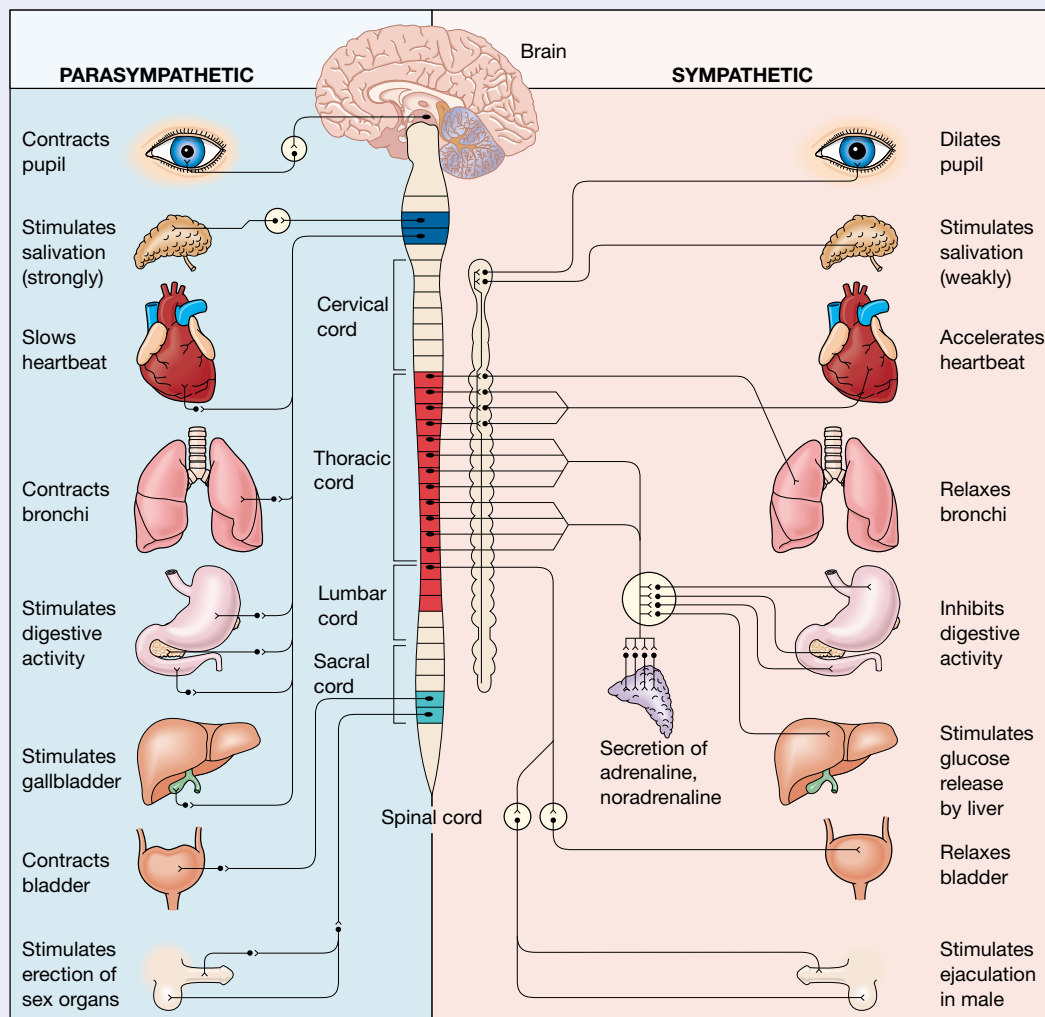
and B cells. T-helper cells promote the release of cytokines; B cells release antibodies that deal with specific pathogens.

## FOCUS ON DISCOVERY 1.2

### The autonomic nervous system

The autonomic nervous system (ANS) innervates the endocrine glands, the heart and the smooth muscles that are found in the walls of the blood vessels, stomach, intestines, kidneys and other organs. This nervous system is itself divided into two parts, the **sympathetic nervous system** and the **parasympathetic nervous system** (see figure 1.12). A simple way to think about these two components of the ANS is that the sympathetic nervous system prepares the body for 'fight or flight' and the parasympathetic nervous system helps 'calm down' the body. Things are not actually that simple, though. The sympathetic portion of the ANS, when energised, accelerates the heartbeat, dilates the pupils, inhibits intestinal activity, increases electrodermal activity (i.e., sweat on the skin) and initiates other smooth muscle and glandular responses that prepare the organism for sudden activity and stress. Division of activities is not quite so clear-cut, however, for it is the parasympathetic system that increases blood flow to the genitals during sexual excitement.

**FIGURE 1.12** The autonomic nervous system



The autonomic nervous system figures prominently in many of the anxiety disorders, such as panic disorder and post-traumatic stress disorder. For example, people with panic disorder tend to misinterpret normal changes in their nervous system, such as shortness of breath after running up a flight of stairs. Instead of attributing this to being out of shape, people with panic disorder may think they are about to have another panic attack. In essence, they come to fear the sensations of their own autonomic nervous system.

#### QUESTIONS

1. Name and describe the two major parts of the autonomic nervous system.
2. Describe the role of the autonomic nervous system in anxiety disorders.

Figure 1.13 shows the components of the immune system. The effects of stress on the immune system are direct and they can happen very early in life. For example, compared to infants of mothers who experienced no stress during pregnancy, the infants of rhesus monkey mothers who were exposed to chronic stress during pregnancy (exposure to loud and unpredictable noises five days a week for one-quarter of their pregnancy) were observed to have emotion regulation difficulties as babies and adolescents that negatively impacted their place in the social group (Coe, Lubach, & Schneider, 1999; Roughton, Schneider, Bromley, & Coe, 1998). In addition, these babies exhibited immune system disturbances that continued into adolescence, including a deficiency of pro-inflammatory cytokines, such as interleukin-6 (Coe, Kramer, Kirschbaum, Netter, & Fuchs, 2002). Interleukin-6 (IL-6) promotes inflammation in response to infection and is importantly linked to human diseases as well as psychological disorders such as major depressive disorder. In addition, exposure to stress also slows the process of wound healing, which relies on the immune system (Kiecolt-Glaser & Glaser, 2002). Across a number of studies, age appears to be a factor — that is, older adults are more likely than younger adults to show a harmful immune response to stress.

Other evidence indicates that stress can trigger the release of cytokines such as interleukin-1 and interleukin-6, as if the body were fighting off an infection (Maier & Watkins, 1998). The link between stress and the immune system has several direct and important implications for overall health. Inflammation and higher levels of IL-6 have been linked to a number of diseases in older adults, such as coronary heart disease, arthritis, multiple myeloma, non-Hodgkin's lymphoma, osteoporosis and type 2 diabetes. Thus, if stress increases the release of IL-6, the impact on health is likely to be negative. In short, the effects of stress on the immune system can be substantial.

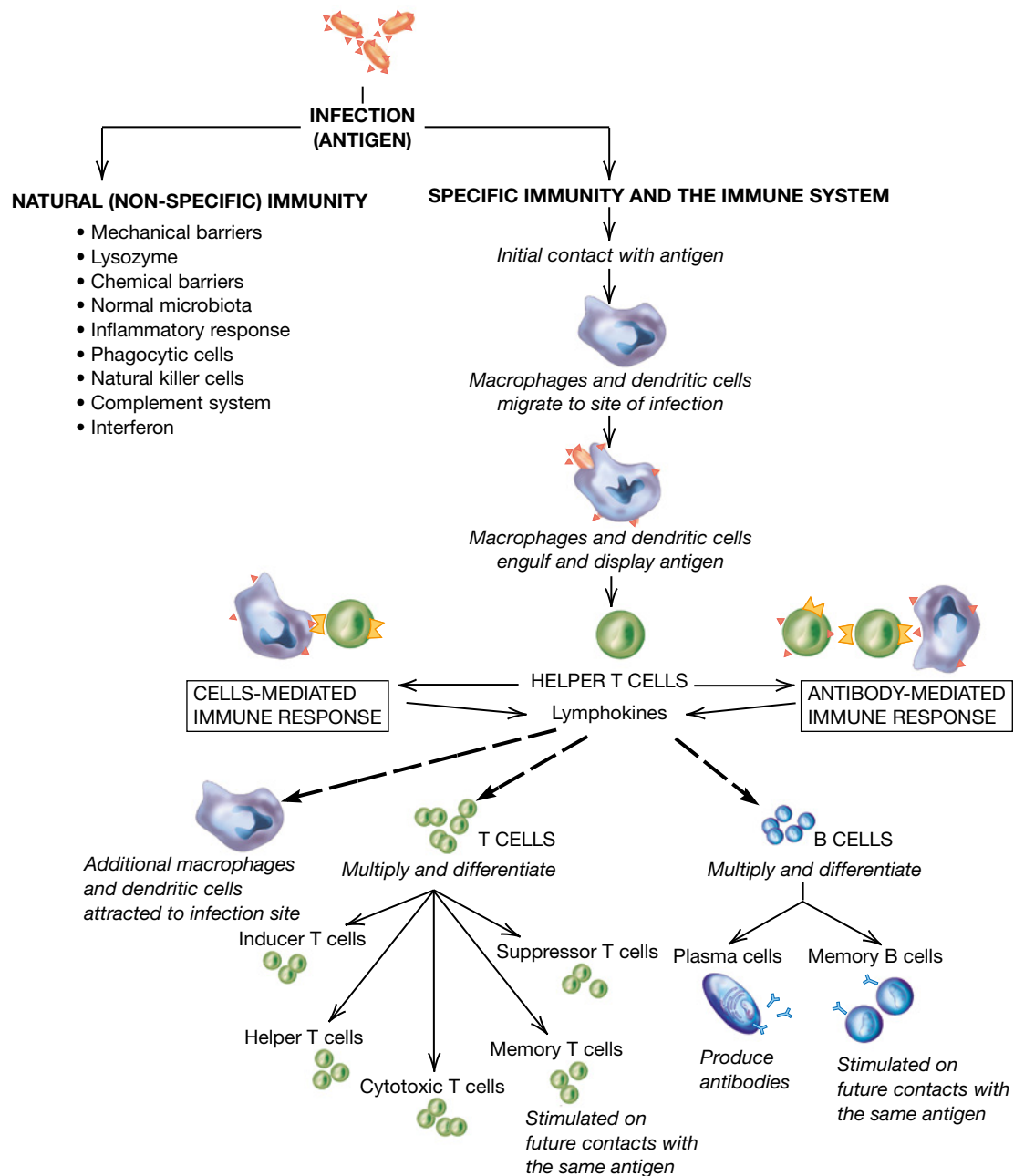
With respect to psychological disorders, recent reviews by Furtado and Katzman (2015a, 2015b) found associations between HPA hyperactivity and both depression and a range of anxiety-related disorders (anxiety, post-traumatic stress and OCD) although there was some inconsistency in findings for the latter disorders. Nonetheless, HPA hyperactivity has been found to decrease in treatment-responsive patients with both medication and psychological treatments. Furthermore, various inflammatory biomarkers are heightened in patients with major depression, lowered in treatment-responsive patients and remain elevated in patients resistant to specific serotonergic medication treatments. Moreover, cytokines are increased in disorders such as post-traumatic stress disorder and OCD. Understanding the psychoneuro-immunological mechanisms of these disorders will help us develop more targeted and effective treatments.

## Neuroscience approaches to treatment

Numerous medications are available that act on psychological symptoms. Antidepressants, such as Prozac, increase neural transmission in neurons that use serotonin as a neurotransmitter by inhibiting the reuptake of serotonin. **Benzodiazepines**, such as Xanax, can be effective in reducing the tension associated with some anxiety disorders, perhaps by stimulating GABA neurons to inhibit other neural systems that create the physical symptoms of anxiety. Antipsychotic drugs, such as Olanzapine, used in the treatment of schizophrenia, reduce the activity of neurons that use dopamine as a neurotransmitter by

blocking their receptors and also impact serotonin. **Stimulants**, such as Ritalin, are often used to treat children with attention-deficit hyperactivity disorder; they operate on several neurotransmitters that help children pay attention.

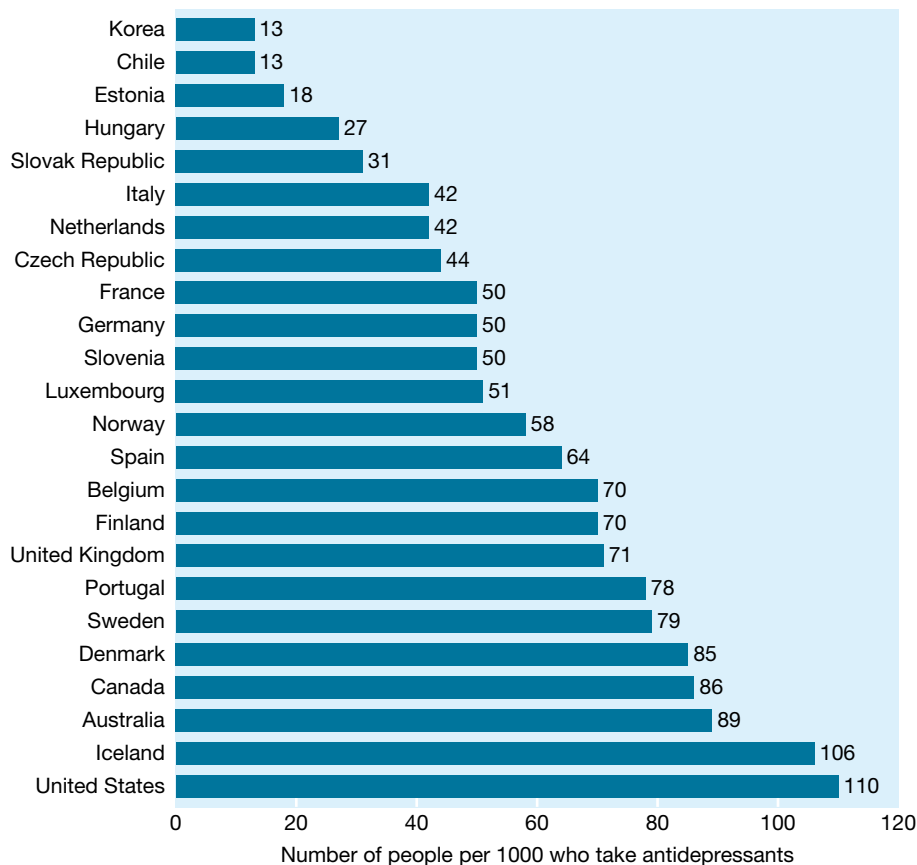
**FIGURE 1.13** Components of the immune system



The use of **psychoactive** drugs such as these has been increasing dramatically worldwide. This is best reflected in the increased patterns of antidepressant use. In its 2015 *Health at a Glance* report, the

Organization for Economic Cooperation and Development (OECD) examined antidepressant use in 25 countries and found that antidepressant use increased in every country between 2000 and 2013. Iceland reported the highest level of consumption of antidepressants in 2013, twice the OECD average, followed by Australia, Portugal and Canada, while Chile, Korea and Estonia reported the lowest consumption levels. The results showed that antidepressant use rose by 46 percent in Germany, and around 20 percent in Spain and Portugal, while one person in ten in Iceland took a daily antidepressant. While the United States was not included in the OECD report, data from the US indicate that 11 percent of individuals over 12 years of age take an antidepressant, making it the highest user of antidepressant medications (see figure 1.14, which presents OECD and US data). Numerous factors, which vary between countries, impact on the level of antidepressant use, including depression **prevalence** rates, how depression is diagnosed, local treatment guidelines, prescribing behaviour by medical and mental health practitioners, pharmaceutical promotional practices and the availability of other treatments, particularly affordable and evidence-based psychological treatments.

**FIGURE 1.14** Global antidepressant users per 1000 people



*Source:* Gould and Friedman (2016).

In Australia, the latest publicly available data on subsidised mental health-related medications from the Australian Bureau of Statistics are for 2011 (see table 1.3). According to that data, antidepressants were the most highly prescribed psychotropic medications with over 1.6 million Australians accessing subsidised antidepressants, followed by anti-anxiety and hypnotic medications with nearly 1 million Australians prescribed one of this group of medications. From the OECD report, the average defined

daily dose (DDD) for antidepressants rose dramatically. In Australian adults, the DDD more than doubled from 2000 to 2013. However, as reviewed by Kyrios (2014), changes in primary care management in Australia via a government initiative called ‘Better Access’ have led to improved accessibility to mental health treatment. General medical practitioners or psychiatrists can administer pharmacological treatment, while a range of professionals including generalist and specialist clinical psychologists can administer reputable evidence-based psychological treatments. As a result of the Better Access initiative, more Australians reported receiving information about mental health conditions and relevant treatment, although there were also increases in unmet needs. Subsequent to improved access to effective evidence-based psychological treatments such as **cognitive-behavioural therapy (CBT)**, there was also a reported decrease in use of antidepressant and anti-anxiety medication (Atlantis, Sullivan, Sartorius, & Almeida, 2012). However, possibly due to workforce shortages and unmet needs, it appears that prescription of psychotropic medications has subsequently increased.

**TABLE 1.3 PBS-subsidised mental health-related medications accessed in 2011**

Type of medication	Number of people	Proportion (%) <sup>(a)</sup>	Average number of prescriptions <sup>(b)</sup>
Antipsychotics	349 900	1.6	8.3
Anxiolytics/Hypnotics and sedatives	921 100	4.3	5.3
Antidepressants	1 678 900	7.8	8.2
Psychostimulants, agents used for ADHD and nootropics	81 400	0.4	7.0
Total persons who had at least one script filled for PBS subsidised mental health-related medication in 2011	2 354 100	10.9	9.4

<sup>(a)</sup> Proportion of the total Australian population.

<sup>(b)</sup> Average number of prescriptions filled for the particular medication in 2011, of people who had at least one prescription filled for the particular medication in 2011.

*Source:* ABS (2011).

Although one might assume that we have learned which neurotransmitters are involved in a disorder and then used that to determine pharmacological treatments, this is not necessarily the case. Rather, the reverse has often happened: a drug is found that influences symptoms and then researchers are inspired to study the neurotransmitters influenced by that drug. However, the evidence linking neurotransmitters as causal factors in psychopathology is not very strong.

It should be noted that a person could hold a neuroscience view about the nature of a disorder and yet recommend psychological intervention. In fact, guidelines for the treatment of depression and anxiety recommend use of CBT as the first line of treatment (see the United Kingdom’s National Institute for Health and Care Excellence, NICE, clinical practice guidance on the appropriate treatment and care of people with specific diseases and conditions: [www.nice.org.uk](http://www.nice.org.uk)). Contemporary scientists and clinicians also appreciate that non-biological interventions can influence brain functioning. For example, **psychotherapy** that teaches a person how to stop performing compulsive rituals, which is an effective and widely used behavioural treatment for OCD, has measurable effects on brain activity (Baxter et al., 2000).

## Evaluating the neuroscience paradigm

Biology gives you a brain. Life turns it into a mind. (Jeffrey Eugenides, *Middlesex*, 2002, p. 479)

Over the past three decades, neuroscientists have made steady progress in elucidating brain-behaviour relationships. Neuroscience research on both the causes and treatment of psychopathology is proceeding

at a great rate, as we will see when we discuss specific disorders in later chapters. Although we view these developments in a positive light, we also want to caution against reductionism.

Reductionism refers to the view that whatever is being studied can and should be reduced to its most basic elements or constituents. In the case of psychological disorders, reductionism happens when scientists try to reduce complex mental and emotional responses to simple biological processes. In its extreme form, reductionism asserts that psychology and psychopathology are ultimately nothing more than biology. The eugenics movement was the outcome of extreme reductionism.

Basic elements, such as individual nerve cells, are organised into more complex structures or systems, such as neural networks or circuits. The properties of these neural circuits cannot be deduced from the properties of the individual nerve cells. The whole is greater than the sum of its parts. A good example is provided by computers. Students writing papers for their courses use word-processing programs like Google Docs, Apple Pages or Microsoft Word. These software programs consist of many levels of code that communicate with the computer. The word-processing program necessarily involves low-level communication with the computer, involving a series of 0s and 1s and even electronics. Yet we don't conceptualise the program in terms of binary digits or electrical impulses. If the spell-checker stopped working, our first place to begin repairs would not be with the computer chips. Instead, we would want a programmer to fix the bug in the program. To be sure, the program could not run without the computer, but the program is more than just the impulses sent by the chips. Furthermore, failing a paper would not be due solely to a problem with the word-processing program, but rather a complex interaction of factors. In the same way, although a complex behaviour like a hallucination necessarily involves the brain and nerve impulses, it is not likely that we can fully capture this by knowing about specific nerve impulses.

Certain phenomena emerge only at certain levels of analysis and will be missed by investigators who focus only on the molecular or cellular level. In the field of psychopathology, problems such as delusional beliefs, dysfunctional attitudes and catastrophising cognitions may well be impossible to explain neurobiologically, even with a detailed understanding of the behaviour of individual neurons (Turkheimer, 1998). The **biopsychosocial framework** highlights the significance of biological, psychological and social factors and their interactions in understanding behaviour. Hence, a comprehensive understanding of psychopathology requires more than consideration of biological factors.

## 1.6 The cognitive–behavioural paradigm

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**LEARNING OUTCOME 1.6** Describe the essentials of the cognitive–behavioural paradigm.

The **cognitive–behavioural paradigm** traces its roots to learning principles and to cognitive science. As we will see, the basic principles from classical and operant conditioning as well as cognitive science have shaped the development of many cognitive–behavioural therapies.

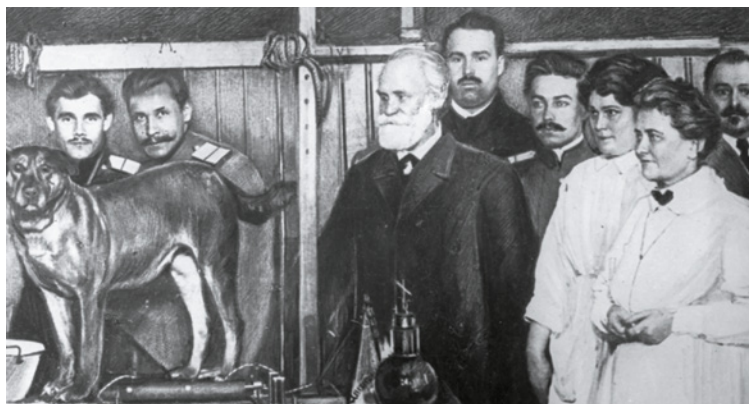
### Influences from behaviour therapy

**Behaviourism** focuses on observable behaviour. Three types of learning influenced the behaviourist approach in the early and middle parts of the twentieth century and these continue to be influential today: classical conditioning, operant conditioning and modelling.

#### Classical conditioning

Around the turn of the twentieth century, the Russian physiologist and Nobel laureate Ivan Pavlov (1849–1936) discovered **classical conditioning**. As part of his study of the digestive system, Pavlov gave a dog meat powder to make it salivate. Before long, the dog began salivating when it saw the person who fed it. As the experiment continued, the dog began to salivate even earlier, when it heard the footsteps of its feeder. Pavlov was intrigued by these findings and decided to study the dog's reactions systematically. In the first of many experiments, a bell was rung behind the dog and then the meat powder was placed in its mouth. After this procedure had been repeated a number of times, the dog began salivating as soon as it heard the bell and before it received the meat powder.

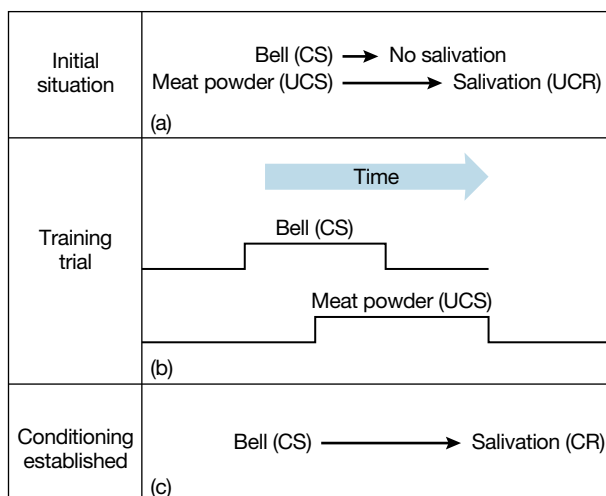
Ivan Pavlov, a Russian physiologist and Nobel laureate, made important contributions to the research and theory of classical conditioning.



In this experiment, because the meat powder automatically elicits salivation with no prior learning, the powder is termed an **unconditioned stimulus (UCS)** and the response of salivation an **unconditioned response (UCR)**. When the offering of meat powder is preceded several times by a neutral stimulus, the ringing of a bell, the sound of the bell alone (the **conditioned stimulus** or **CS**) is able to elicit the salivary response (the **conditioned response** or **CR**) (see figure 1.15). As the number of paired presentations of the bell and the meat powder increases, the number of salivations elicited by the bell alone increases. What happens to an established CR if the CS is no longer followed by the UCS — for example, if repeated soundings of the bell are not followed by meat powder? The answer is that fewer and fewer CRs (salivations) are elicited and the CR gradually disappears. This is termed **extinction**.

**FIGURE 1.15**

The process of classical conditioning. (a) Before learning, the meat powder (UCS) elicits salivation (UCR), but the bell (CS) does not. (b) A training or learning trial consists of presentations of the CS, followed closely by the UCS. (c) Classical conditioning has been accomplished when the previously neutral bell elicits salivation.



Extinction is thought to be an associative way to (a) ‘unlearn’ the UCS-CS associations; (b) ‘learn’ an inhibitory association that masks the original excitatory association or (c) experience a non-associative mechanism through **habituation** — for instance, when an animal has been exposed to a stimulus repeatedly with no harmful consequence, it can merely stop responding. Laboratory work by Myers and

Davis (2007) on fear extinction in rats supports multiple mechanisms contingent on the timing and conditions of extinction.

Classical conditioning notions have been applied to treating humans and can even instil pathological fear. A famous experiment conducted by John Watson and Rosalie Rayner (1920) — now considered to be ethically questionable — involved introducing a white rat to an 11-month-old boy, Little Albert. The boy showed no fear of the animal and appeared to want to play with it. But whenever the boy reached for the rat, the experimenter made a loud noise (the UCS) by striking a steel bar behind Albert's head. This caused Little Albert great fright (the UCR). After five such experiences, Albert became very frightened (the CR) by the sight of the white rat, even when the steel bar was not struck. The fear initially associated with the loud noise had come to be elicited by the previously neutral stimulus, the white rat (now the CS). This study suggests a possible relationship between classical conditioning and the development of certain disorders, in this instance a phobia. It is important to note that this type of study could never be done today because it breaches ethical standards.

In any case, the study of extinction learning has been extended to humans and generally supports comparable findings from animal and human research with a few exceptions (Hermans, Craske, Mineka, & Lovibond, 2006). Hermans et al. (2006) concluded that extinction does not involve unlearning and that inhibitors can lead to a partial recovery of the seemingly 'extinguished' fear responses. This has important implications for psychological treatment, which is covered in a later section of this chapter.

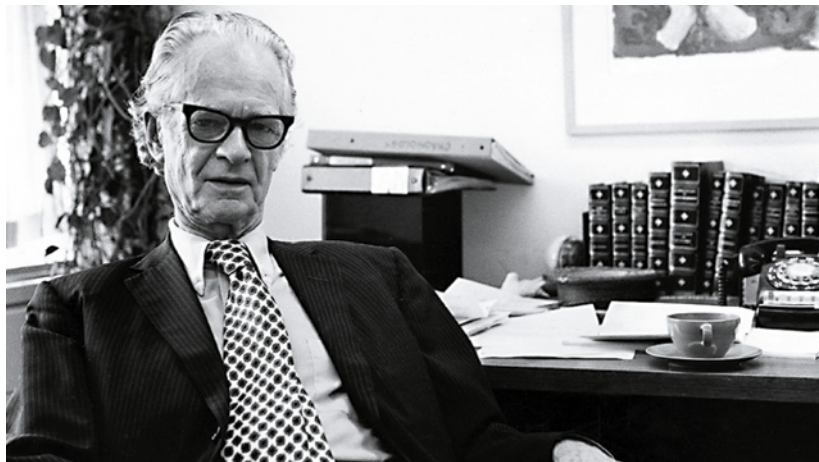
### Operant conditioning

In the 1890s, Edward Thorndike (1874–1949) began work that led to the discovery of another type of learning. Rather than focusing on associations between stimuli, as Pavlov did, Thorndike studied the effects of their *consequences* on behaviour. Thorndike formulated an extremely important principle, the **law of effect**: behaviour that is followed by consequences satisfying to the organism will be repeated and behaviour that is followed by noxious or unpleasant consequences will be discouraged.

Later, B. F. Skinner (1904–1990) introduced the concept of **operant conditioning**, so called because it applies to behaviour that operates on the environment. Renaming Thorndike's 'law of effect' the 'principle of reinforcement', Skinner distinguished two types of reinforcement. **Positive reinforcement** refers to the strengthening of a tendency to respond by virtue of the presentation of a pleasant event, called a positive reinforcer. For example, a water-deprived pigeon will tend to repeat behaviours (operants) that are followed by the availability of water. **Negative reinforcement** also strengthens a response, but it does so via the removal of an aversive event, such as the cessation of electric shock.

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B. F. Skinner originated the study of operant conditioning and the extension of this approach to education, psychotherapy and society as a whole.



Operant conditioning principles may contribute to the persistence of aggressive behaviour, a key feature of conduct disorder (see the chapter on disorders of childhood). Aggression is often rewarded, as when one child hits another to secure the possession of a toy (getting the toy is the reinforcer). Parents may also unwittingly reinforce aggression by giving in when their child becomes angry or threatens violence to achieve some goal, such as staying up late to watch TV. Operant conditioning is also useful in understanding the persistence of anxiety disorders such as phobias (see the chapter on anxiety, obsessive-compulsive and trauma-related disorders). For instance, phobias are frequently rewarded by the attention that someone receives when they are fearful in the presence of the phobic stimulus (e.g., a dog). Escaping from a phobic stimulus will remove noxious emotional experiences such as panic, thus reinforcing the phobia.

## Modelling

Learning often goes on even in the absence of reinforcers. We all learn by watching and imitating others, a process called **modelling**. In the 1960s, experimental work demonstrated that witnessing someone perform certain activities can increase or decrease diverse kinds of behaviour, such as sharing, aggression and fear. For example, Bandura and Menlove (1968) used a modelling treatment to reduce fear of dogs in children. After witnessing a fearless model engage in various activities with a dog, initially fearful children showed an increased willingness to approach and touch a dog. Children of parents with phobias or substance abuse problems may acquire similar behaviour patterns, in part through observation.

## The emergence of behaviour therapy

Subsequent to understanding contingencies that act on behaviour, **behaviour therapy** emerged in the 1950s to alter clinical problems. In its initial form, this therapy applied procedures based on classical and operant conditioning. Sometimes the term *behaviour modification* is used and therapists who use operant conditioning as a treatment often prefer that term. Behaviour therapy was an attempt to change behaviour, thoughts and feelings by applying, in a clinical context, the methods used and the discoveries made by experimental psychologists.

Generally, problem behaviour is thought to be reinforced by four possible consequences: getting attention, escaping from tasks, generating sensory feedback (such as results from the hand flapping often seen in children with autism spectrum disorder) and gaining access to desirable things or situations (Carr et al., 1994). Once the source of reinforcement has been identified, treatment is then tailored to alter the consequences of the problem behaviour. One successful example of operant conditioning is **behavioural activation (BA) therapy** of depression (Dimidjian, Barrera, Martell, Muñoz, & Lewinsohn, 2011), which involves helping a person engage in tasks that provide an opportunity for positive reinforcement. Operant techniques such as systematically rewarding desirable behaviour and extinguishing undesirable behaviour have been particularly successful in the treatment of many childhood problems (Kazdin & Weisz, 1998). For example, having established that getting attention reinforced a problem childhood behaviour, parents and teachers might be advised to ignore it. Alternatively, the problem behaviour could be followed by **time-out** — the child could be sent for a period of time to a location such as a quiet room where positive reinforcers are not available. Today, time-out is a commonly used parenting technique for children who exhibit a problematic behaviour of some sort.

Once contingencies shape a behaviour, a key goal is to maintain the effects of treatment. If a therapist or a teacher has been providing reinforcement, one might not expect this person to keep providing reinforcement forever. This issue has been addressed in several ways. Because laboratory findings indicate that intermittent reinforcement — rewarding a response only a portion of the times it appears — makes a new behaviour more enduring, many operant programs move away from continuous schedules of reinforcement once desired behaviour is occurring regularly. For example, if a teacher has succeeded in helping a disruptive child spend more time sitting by praising the child generously for each maths problem finished while seated, the teacher will gradually reward the child for every other success and ultimately only infrequently.

One important behaviour therapy technique that is still used to treat phobias and anxiety today is called **systematic desensitisation**. Developed by Joseph Wolpe in 1958, it includes *two components*: (1) deep muscle relaxation and (2) gradual **exposure** to a list of feared situations, starting with those that arouse minimal anxiety and progressing to those that are the most frightening. Wolpe (1954) hypothesised that a state or response opposite to anxiety is substituted for anxiety as the person is exposed gradually to stronger and stronger doses of what he or she fears. He suggested that reciprocal inhibition, 'the complete or partial suppression of the anxiety responses as a consequence of the simultaneous evocation of other responses physiologically antagonistic to anxiety' (Wolpe, 1954, p. 205), was a fundamental psychotherapeutic mechanism. In other words, one cannot be anxious and relaxed at the same time, and training people to relax when exposed to gradually increasing anxiety in the presence of a phobic stimulus will help them overcome their phobia. While systematic desensitisation remains a part of some current forms of cognitive-behavioural therapy, there is growing appreciation of greater complexity beyond reciprocal inhibition in understanding therapeutic mechanisms.

In more recent times, the exposure component has become more influential. The basic idea is that anxiety will extinguish if the person can face a feared object or situation long enough with no actual harm occurring. Sometimes this exposure can be conducted **in vivo** — that is, in real-life situations. For example, if someone has a fear of flying, you might have him or her take an actual flight. At times, exposure cannot be conducted in real life, so *imaginal exposure* will be used to address fears, such as rape, trauma or contamination. In other situations, both types of exposure are used. Graded exposure has most often been used in the treatment of anxiety disorders whereby affected individuals are exposed to situations or objects associated with gradually increasing levels of anxiety. Progress to exposure to a situation or object associated with the next level of anxiety or distress is contingent on the affected individual coping with the previous level. In the years since exposure treatments were first developed, much has been learned about them. For instance, even though relaxation training helps people experience less arousal when they first face the feared stimulus, there is little evidence that such training is required for good outcomes. As a result, it has been possible to develop briefer psychological treatments that do not include relaxation training.

The utility of a habituation-based understanding of exposure has recently been questioned and an **inhibitory learning** model supported (Craske, 2014). Inhibitory learning is learning that inhibits previous learning. Research has shown people with anxiety disorders demonstrate deficits in inhibitory learning. Craske has highlighted that, while habituation is beneficial, it may not have desired long-term effects. She considers that targeting such processes by using a combination of behavioural and cognitive strategies is likely to improve enduring outcomes following exposure (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014). Acquisition of behaviours that are incompatible with customary habits

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Time-out is a behavioural therapy technique based on operant conditioning; the consequence for misbehaviour is removal to an environment with no positive reinforcers.



and the development of exposure situations that better match natural events will maximise outcomes. Furthermore, it's important for anxious individuals to test their new strategies, refine them and facilitate their generalisation to other threatening settings.

Modelling has also been included in behaviour therapy. For example, people reduced their fear of animals such as snakes by viewing both live and filmed encounters in which other people gradually approached and successfully handled snakes (Bandura, Blanchard, & Ritter, 1969). Fears of surgery and dental work have also been treated in a similar manner (Melamed & Siegel, 1975).

As influential as these behaviour therapy techniques have been, behaviourism and behaviour therapy have been criticised for minimising the importance of two important factors: thinking and feeling. In other words, the way we think and feel about things undoubtedly influences our behaviour. These limitations of behavioural points of view led some behavioural researchers and clinicians to include cognitive and emotion variables in their conceptualisations of psychopathology and therapy.

## Cognition

### The importance of cognition

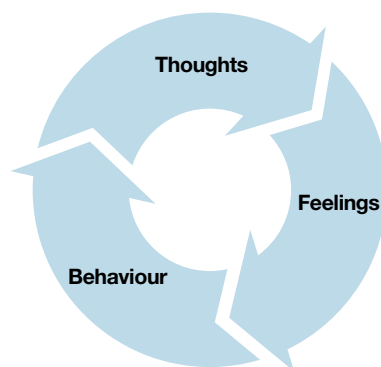
Human beings don't just behave; they think and feel, too! Early behaviour theories did not leave much room for **cognition** and emotion. Beginning in the 1960s, the study of cognition began to gain prominence. Researchers and clinicians realised that the ways in which people think about situations influences behaviour in dramatic ways. For example, walking into a room of strangers can elicit thoughts such as 'Great! I am so excited to meet all sorts of new and interesting people' or 'I do not know any of these people and I am going to look and sound like a complete fool!' A person who has the first thought is likely to join a group of people enthusiastically and join in the conversation. The person who has the second thought, however, is likely to turn right around and leave the room.

### Framework of cognitive therapy

Cognitive theory emphasises that the way in which people construe themselves, the world and the future is a major determinant of psychological disorders. In cognitive approaches to therapy, the therapist typically begins by helping clients become more aware of their maladaptive thoughts. By changing cognition, therapists aim to help people change their maladaptive feelings, behaviours and symptoms.

The roots of cognitive therapy include Aaron Beck's **cognitive therapy** and Albert Ellis's **rational-emotive behaviour therapy (REBT)**.

**FIGURE 1.16** Beck's (1967) cognitive theory — relationship between thoughts, feelings and behaviour



Ellis's (1913–2007) principal thesis was that sustained emotional reactions are caused by internal sentences that people repeat to themselves; these self-statements reflect sometimes unspoken assumptions

— irrational beliefs — about what is necessary to lead a meaningful life. In Ellis's REBT (Ellis, 1993, 1995), the aim is to eliminate self-defeating beliefs. A person with depression, for example, may conclude on the basis of work or relationship challenges, 'I am worthless that no-one will ever care if I'm gone.' Ellis proposed that people interpret what is happening around them on the basis of set beliefs (e.g., 'I must be perfect'), that these interpretations cause emotional turmoil and that a therapist's attention should be focused on these beliefs rather than on historical causes or, indeed, on overt behaviour.

Ellis later (1991; Kendall et al., 1995) shifted from a cataloguing of specific irrational beliefs to the more general concept of 'demandingness', that is, the 'musts' or 'shoulds' that people impose on themselves and on others. Ellis hypothesised that it is this unrealistic, unproductive demand that creates the kind of emotional distress and behavioural dysfunction that bring people to therapists.

Aaron Beck's contribution to cognitive therapy and theory has been particularly influential in the treatment of an ever-increasing range of psychological disorders, although his initial focus was on depression.

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Aaron Beck developed a cognitive-theory of depression and a cognitive-behavioural therapy for people with depression.



Like Ellis, Beck (1967) also proposed that our thoughts determine our feelings and our behaviour (see figure 1.16). Irrational and unrealistic thoughts are proposed as leading to unconstructive or negative misinterpretations of situations or about the world, one's self worth and the future, which lead to distress and unhelpful behaviours or reactions, not only resulting in personal impairment but also reinforcing the distorted thinking. Negative emotional distress, in turn, leads to biased cognitive processing and easier access to further negative interpretations that, in turn, perpetuate the emotional distress. Hence, the cognitive approach proposes that psychopathology is caused by faulty cognitive content (irrational beliefs about the world, self and future) and distorted information processing (cognitive distortions).

From the opening scenario, Emma and Ahmed demonstrated significant distress and impairment based on their perceptions of the understandable ambivalence they were experiencing as a couple about having a child. Emma's fears were based on unacknowledged expectations that she would genetically pass on her mental health problems to her children. Ahmed, on the other hand, was concerned about fulfilling others' expectations about having children and his worries about whether he could cope with future parenthood. Once the unhelpful perceptions of the situation were resolved, Emma and Ahmed were able to resolve their distress.

## Cognitive science

*Cognition* is a term that groups together the mental processes of perceiving, recognising, conceiving, judging and reasoning. Cognitive science focuses on how people (and animals) structure their experiences, how they make sense of them and how they relate their current experiences to past ones that have been stored in memory. While at any given moment we are bombarded by far more stimuli than we can possibly respond to, we filter this overwhelming input, put it into words or images, form hypotheses and arrive at an interpretation of what is out there. Cognitive research has provided some support for the way in which we do all this and the importance of cognitive processes in the aetiology and maintenance of psychopathology. Cognitive scientists regard people as active interpreters of a situation, with people's past knowledge imposing a perceptual funnel on the experience. A person fits new information into an organised network of already accumulated knowledge, often referred to as a **schema** or cognitive set (Neisser, 1976). New information may fit the schema; if not, the person reorganises the schema to fit the information or construes the information in such a way as to fit the schema. The following situation illustrates how a schema may alter the way in which information is processed and remembered.

The man stood before the mirror and combed his hair. He checked his face carefully for any places he might have missed shaving and then put on the conservative tie he had decided to wear. At breakfast, he studied the newspaper carefully and, over coffee, discussed the possibility of buying a new washing machine with his wife. Then he made several phone calls. As he was leaving the house he thought about the fact that his children would probably want to go to that private camp again this summer. When the car didn't start, he got out, slammed the door and walked down to the bus stop in a very angry mood. Now he would be late. (Bransford & Johnson, 1973, p. 415)

Now read the excerpt again, but add the word *unemployed* before the word *man*. Now read it a third time, substituting *investment banker* for *man*. Notice how differently you understand the passage. Ask yourself what parts of the newspaper these men read. If you were asked on a questionnaire to recall this information and you no longer had access to the excerpt, you might answer 'the want ads' for the unemployed man and 'the financial pages' for the investment banker. Since the passage does not specify which part of the paper was read, these answers could be wrong, but in each instance the error would have been a meaningful, predictable one.

Other important contributions from cognitive science include the study of attention. As we will see, people with disorders as diverse as anxiety disorders, mood disorders and schizophrenia have problems with attention. For example, individuals with anxiety disorders tend to focus their attention on threatening or anxiety-producing events or situations in the environment. People with schizophrenia have a hard time concentrating their attention for a period of time.

One of the ways in which researchers have studied attention is with the *Stroop task*. In this task, the participant sees a set of colour names printed in inks of *different* colours and must name the ink colour of each word as rapidly as possible (see figure 1.17). To do this, participants have to resist the natural impulse to say the printed word. For example, a participant might see the word *blue* written in green ink. The participant is instructed to name the ink colour (green) as fast as possible without making mistakes (saying the word *blue*). It is difficult to say *green* and 'inhibit' the more natural tendency to say *blue*. Interference, measured as a lengthening of response time, occurs because the words are more 'attention grabbing' than the ink colour.

**FIGURE 1.17** In the Stroop task, participants must name the colour of the ink instead of reading the words.

Black	Pink
Red	White
Blue	Red
Green	Black
Yellow	Purple
Blue	Green
Red	Blue
White	Yellow

The Stroop task has been modified to focus on emotion rather than colours. In this *emotion Stroop task*, participants are still instructed to name the colour of the ink rather than say the word. However, the list of words now contains emotion words instead of colour words. So, for example, words such as *threat*, *danger*, *happy* or *anxious* are written in different ink colours. In such an emotion Stroop task, individuals with anxiety disorders find that some of the emotion words are so attention grabbing that the impulse to say the word is especially strong. As in the original Stroop task, the more attention grabbing the word is, the more interference and the slower the response. Research has shown that people with anxiety disorders show more interference for threatening words (i.e., they say these words more slowly) than for non-threatening words; this is used as evidence of an attention bias towards threatening information.

Of course, the concepts of schema and attention are related to each other. If a person has a particular cognitive set or schema about the world (e.g., the world is dangerous), that person may be more likely to pay attention to threatening or dangerous things in the environment. Furthermore, this person may be more likely to interpret ambiguous things in the environment as threatening. For example, seeing a stranger standing on a front porch may be interpreted as a sign of danger to someone with such a 'danger' schema. For someone without such a schema, this person may be viewed simply as the person who lives in that house.

Researchers have become interested in the inter-relationships between genetic predispositions, psychopathology and attentional and attributional processes. For instance, Lau and Eley (2008) used longitudinal twin and sibling data to investigate attributional style as a marker of genetic risk for adolescent depression. They found that attributional style was moderately heritable at both time points over an average interval of 25 months with genetic links between concurrent and prospective depression impacted by age, gender and levels of stress. Furthermore, in a study of monozygotic and dizygotic 10-year-old twin pairs, Lau, Hilbert, Goodman, Gregory, Pine, Viding, and Eley (2012) found that recognition of socially threatening faces was moderately heritable, while avoidant responses were heavily influenced by the non-shared environment. Hence, we see further evidence of complexity in the aetiology of psychopathology with cognitive factors interacting with genetic, experiential and other factors to impact on outcomes. Various aspects of cognitive functioning have been the focus of research interest.

### The role of the unconscious

While cognitive explanations are central in the search for the causes of psychopathology, new methods have been developed to measure those biases and to change them. These approaches have been useful for understanding and treating a range of disorders, particularly anxiety-based disorders (see MacLeod & Clarke, 2015). The distinction between conscious and unconscious **cognitive biases** has been of particular interest.

As far back as Sigmund Freud (discussed earlier in this chapter), much of human behaviour was presumed to be unconscious or outside the awareness of the individual. Later followers of Freud continued to emphasise the role of the unconscious in human behaviour and psychopathology, but the way in which the unconscious has been discussed and even empirically studied has changed over the years.

The unconscious has been a hot topic of study among cognitive psychologists for over 30 years. For example, in one study, participants were presented with different shapes for 1 millisecond (one-thousandth of a second) (Kunst-Wilson & Zajonc, 1980). Later they showed virtually no ability to recognise the shapes they had seen, but when they rated how much they liked the shapes, they preferred the

ones they had been shown to other ones. We know that familiarity affects judgements of stimuli; people tend to like familiar stimuli more than unfamiliar ones. This study indicates that some aspects of the stimuli must have been absorbed, even though participants said that they did not recognise the shapes.

Cognitive neuroscientists have more recently explored how the brain supports behaviour that is outside conscious awareness. For example, the concept of *implicit memory* refers to the idea that a person can, without being aware of it, be influenced by prior learning. A person may be shown a list of words so quickly that he or she cannot identify the words. Later, the person will be able to recall those words even though the words were not consciously perceived during the rapid initial presentation. Thus, a memory is formed implicitly (i.e., without conscious awareness). Implicit memory paradigms have been adopted by psychopathology researchers, who have found, for example, that people with social anxiety and depression have trouble with these tasks (Amir, Foa, & Coles, 1998; Watkins, 2002).

Contemporary studies of the unconscious, such as studies of implicit memory, are a long way from Freud's original theorising about the unconscious. For cognitive neuroscientists, the unconscious reflects the incredible efficiency and automaticity of the brain. That is, there are simply too many things going on around us all the time for us to be aware of everything. Thus, our brains have developed the capacity to register information for later use even if we are not aware of it.

## Self-schemas

I have made the self-concept a central feature of my writing and treatment approach for more than five decades — it is a unifying feature in depression, positive and negative symptoms of schizophrenia and personality disorders, to name a few. The notion that we have something in us that expresses itself in so many ways is puzzling, yet elegant and exciting ... When psychological disorders are understood as disorders of self, clinicians can apply a fresh perspective towards treatment. Aaron T Beck (in Kyrios et al., 2016, p. x)

More recently, there has also been increasing interest in negative **self-schemas**, proposed by Beck to be an important component of the cognitive theory of psychopathology (see Kyrios et al., 2016). Negative self-schemas denote a set of implicit and explicit beliefs and expectations about oneself that are unrealistic, unconstructive and negative. Beck proposed that individuals susceptible to depression develop such schemas during their childhood; however, cognitive therapy examines how such self-constructs manifest in day-to-day life, whereas earlier forms of psychotherapy based on Freudian psychology (e.g., psychoanalysis) focused on early childhood experiences and unconscious intrapersonal conflicts. Self-constructs are discussed in greater detail in a following section.

## Cognitive-behavioural therapy

Cognitive-behavioural therapy (CBT), which incorporates cognitive therapy (CT) and behaviour therapy (BT), aims to help people increase their awareness of their irrational thinking and the behavioural repertoires that lead to emotional distress and support the maladaptive thinking patterns. CT and its associated behavioural interventions, usually comprising exposure-based and contingency-based strategies, facilitate the development of alternative thinking styles and behaviours, which impact on emotional functioning and functional impairment.

### Cognitive therapy (CT)

Cognitive therapists pay attention to private events — thoughts, perceptions, judgements, self-statements and even tacit (unconscious) assumptions — and have studied and manipulated these processes in their attempts to understand and modify overt and covert disturbed behaviour. **Cognitive restructuring** is a general term for changing a pattern of thought. People with depression may not realise how often they think self-critically and those with anxiety disorders may not realise that they tend to be overly sensitive to possible threats in the world. Therapists propose that people can change their feelings, behaviours and symptoms by changing their cognitions. The therapist begins by tracking the daily thoughts a person experiences but then moves to understanding more about core cognitive biases and schemata that might shape those daily negative thoughts. Tracking of unhelpful thoughts and practising more helpful behaviours

and thinking styles are core components of CBT, which are incorporated into homework assignments so that ‘therapy’ is taken out of the session with the therapist and into the client’s daily life. The importance of completing homework exercises has been supported by numerous studies. However, a recent review of the homework literature concluded that both quantity and quality of homework compliance were related.

## Beck’s cognitive therapy

Beck’s therapy, which has been adapted for a range of disorders in addition to depression, is a collaborative treatment between a therapist and the person seeking treatment. When a person with depression expresses feelings that nothing ever goes right, for example, the therapist offers counterexamples, pointing out how the person has overlooked favourable happenings. The general goal of Beck’s therapy is to provide people with experiences, both inside and outside the therapy room, that will alter their negative schemas, enabling them to have hope rather than despair.

### CLINICAL CASE

#### An example of Beck’s cognitive therapy

The following examples illustrate ways of beginning to help a person change negative cognitions.

**Therapist:** You said that you feel like a failure since Bill left you. How would you define ‘failure’?

**Patient:** Well, the marriage didn’t work out.

**Therapist:** So, you believe that the marriage didn’t work out because you, as a person, are a failure?

**Patient:** If I had been successful, then he would still be with me.

**Therapist:** So, would we conclude that we can say, ‘People whose marriages don’t work out are failures’?

**Patient:** No, I guess I wouldn’t go that far.

**Therapist:** Why not? Should we have one definition of failure for you and another for everyone else?

People who define *failure* as less than ‘extraordinarily successful’ can see that their definitions are polarised in all-or-nothing terms — that is, ‘complete success’ versus ‘complete failure’. A variation on this technique is to ask the patient how others would define ‘success’ or ‘failure’.

**Therapist:** You can see that your definition of failure is quite different from the way other people might see it. Few people would say that a person who is divorced is a failure. Let’s focus on the positive end right now. How would most people define ‘success’ in a person?

**Patient:** Well, they might say that someone has success when they accomplish some of their goals.

**Therapist:** OK. So, would we say that if someone accomplishes some goals they have success?

**Patient:** Right.

**Therapist:** Would we also say that people can have different degrees of success? Some people accomplish more goals than others?

**Patient:** That sounds right.

**Therapist:** So, if we applied this to you, would we say that you have accomplished some of your goals in life?

**Patient:** Yes, I did graduate from college and I have been working for the past six years. I’ve been busy raising Ted — he had some medical problems a couple of years ago, but I got the right doctors for him.

**Therapist:** So, would we call these some successful behaviors on your part?

**Patient:** Right, I’ve had some successes.

**Therapist:** Is there a contradiction, then, in your thinking — calling yourself a ‘failure’ but saying that you have had several successes?

**Patient:** Yes, that doesn’t make sense, does it?

*Source:* Leahy, 2003, pp. 38–39.



## QUESTIONS

1. Identify one irrational belief and one distorted thinking style that could have led to the emotional problems.
2. What is more important in resolving distress, the acknowledgement of the negative thinking or its replacement with more rational thinking?

## RESEARCH EXAMPLE

### Evaluating cognitive-behavioural therapy

CBT, BT and CT have been found to be effective across a range of disorders. In a 40-year retrospective evaluating the current state of cognitive therapy, Beck (2005) outlined various reviews comparing CBT and CT against control conditions concluding, along with a more recent review (Butler, Chapman, Foreman, & Beck, 2006), that they were very effective in the treatment of mood and anxiety-based disorders. Exposure-based behaviour therapy did equally well in the treatment of adult depression and OCD. CBT was also found to be superior to antidepressant medication for adult depression in some but not all studies, although some studies have reported equal effectiveness at post-treatment but better outcomes for CT with respect to relapse prevention (DeRubeis et al., 2005). Severity of depression did not appear to make a difference to the superiority of CT over antidepressants, while more recent studies of unpublished data found that antidepressants were not effective at all except for very severely depressed patients (Kirsh et al., 2008). In addition, there were moderate effects of CBT for marital distress, anger, childhood somatic disorders and chronic pain, while small effects were found in sexual offenders. Large effects from uncontrolled studies were found for schizophrenia and bulimia nervosa. However, more recent reviews of the efficacy of CBT for eating disorders reached a more circumspect conclusion regarding the effectiveness of CBT for anorexia nervosa (Galsworthy-Francis & Allan, 2014), although it was considered a worthy addition to other equally effective interventions such as dietary counselling, non-specific supportive management, interpersonal therapy and behavioural family therapy (described in a following section).

With respect to severe mental illnesses such as schizophrenia and chronic and/or treatment-resistant mood disorders, recent reviews have indicated the effectiveness of CBT, especially as an adjunct to medication treatments and particularly in effecting improvements in positive symptoms. See a later chapter for a description of the symptoms of schizophrenia (Thase, Kingdon, & Turkington, 2014). Of great optimism is the finding that patients with medication-resistant syndromes also respond to CBT and that improvements are maintained at follow-up.

A breakout series of studies from Australia examining early interventions found that early detection and intervention which includes cognitive therapy is effective in preventing or delaying a first psychotic episode in people at ultra-high risk of developing psychosis (McGorry et al., 2002). As reviewed by Van der Gaag et al. (2013), these results are supported by international studies. Hence, recent clinical practice guidelines by the Royal Australian and New Zealand College of Psychiatrists for the management of schizophrenia and related disorders have supported the integration of cognitive therapy into early intervention programs (Galletly et al., 2016).

CBT has also been found to have large effects in helping people with chronic medical conditions (e.g., diabetes, **cardiovascular disease**, hypertension, cancer, headaches and migraines, chronic pain conditions and irritable bowel syndrome), either on its own or when combined with lifestyle changes. In some instances, cognitive and behavioural strategies, combined with lifestyle interventions, can prevent the onset of chronic medical conditions. For instance, Kyrios et al. (2009) reported findings from a randomised control study where 290 Australian participants recruited from primary care who had pre-diabetes, a condition that predates the onset of type 2 diabetes, participated in a lifestyle z that focused on improving knowledge, motivation, management of lifestyle factors, stress reduction and beliefs about self and diabetes. Forty-two percent of people with pre-diabetes in the intervention group changed to a diagnosis of 'no diabetes' in comparison with 26 percent of the wait control group; in other words, those people with pre-diabetes in the HLC groups were 1.6 times more likely to move to a no-diabetes diagnosis than those who received standard care, over the time period of the study.

An important feature of CBT is its suitability for interventions that can be delivered online or via other digital means (e.g., smartphone apps) in either self-help or therapist-assisted formats. Enormous advances in the online dissemination of CBT have been made in recent times, with Australia a leader in the development of such e-therapies and their integration into the national mental health system (see figure 1.18 from the Special Edition of the Australian Psychological Society's July 2014 *InPsych*, which summarises Australian advances in this area).

As a large body of research now supports the efficacy of e-mental health resources (eMHR) in assessing and treating mental health disorders, it is no surprise that mental health policies are being greatly influenced by the digital revolution (see Reynolds, Griffiths, Cunningham, Bennett, & Bennett, 2015). Recent advances in eMHRs have facilitated potential changes to the way in which individual professionals practise, and mental health facilities organise, services. Furthermore, eMHRs can be used to train, cue and support practitioners, particularly with respect to maintaining fidelity of CBT. Such progress is supported by ever-increasing digital access and improving levels of mental health literacy in the community. While eMHRs are most commonly integrated into early levels of stepped care systems, mental health professionals are also developing specific digital competencies to integrate use of relevant eMHRs into their own practice to facilitate more efficient and cost-effective mental health treatment and greater access to specialised skills for people living in remote geographic locations.

The Australian government, the Australian Psychological Society (APS) and Australian researchers have taken leadership in the development of online treatments for mental health problems, the provision of online services and the development of mental health policies to increase public access to CBT via digital means. Mindhealthconnect ([www.mindhealthconnect.org.au](http://www.mindhealthconnect.org.au)) is an Australian government portal that brings together Australia's leading online mental health providers and allows people to find trustworthy mental health information. The APS has developed a guide to assist psychologists in using eMHRs (see the associated resources for this chapter).

### QUESTIONS

1. Which disorders have been best serviced by the advent of cognitive-behavioural therapies?
2. For which disorders do we still have some significant progress to make with respect to cognitive-behavioural treatments?
3. What advantages and disadvantages can you identify in disseminating psychological treatments via the internet or other digital means?

**FIGURE 1.18** Online and remote psychological treatment services supported by the Australian government's e-mental health strategy

### Online and remote psychological treatment services in Australia

- The Australian National University hosts the *e-hub* Self-Help Programs for Mental Health and Wellbeing ([www.ehub.anu.edu.au/welcome.php](http://www.ehub.anu.edu.au/welcome.php)), a suite of self-help web-based supports for people with anxiety and depression, including: *MoodGym* ([www.moodgym.anu.edu.au](http://www.moodgym.anu.edu.au)), a free self-help program instructing people vulnerable to depression and anxiety in CBT skills; and *eCouch* ([www.ecouch.anu.edu.au](http://www.ecouch.anu.edu.au)), delivering interactive self-help programs offered free of charge with resources for tracking progress and client experiences, and information and strategies for dealing with depression, anxiety, relationship breakdown, and loss and grief.
- The Black Dog Institute is an educational, research, clinical and community-oriented centre funded to offer *myCompass* ([www.mycompass.org.au](http://www.mycompass.org.au)), an interactive self-help service promoting resilience and wellbeing and *BITEBACK* ([www.biteback.org.au](http://www.biteback.org.au)), an interactive website for young people. *myCompass* allows clients to track their moods, diarise their experiences, and view information and tips for managing their mental health. *myCompass* also includes modules that aim to help people manage mild-to-moderate stress, anxiety and depression. *BITEBACK* uses strategies more aligned to the preferences and experiences of youth such as social networking, meditation and mindfulness exercises, text-based and photo blogging, and competitions.
- Boystown *Kids Helpline* ([www.kidshelp.com.au/teens](http://www.kidshelp.com.au/teens)) offers a helpline, web counselling services allowing synchronous one-on-one, real time counseling with a counsellor via the internet, and asynchronous email contact with a counsellor.

- The Clinical Research Unit of Anxiety and Depression (CRUfAD) at St Vincent's Hospital, Sydney maintains the *This Way Up Clinic* (<https://thiswayup.org.au>), a suite of internet-based courses for people with anxiety and depression. Clinicians (e.g., general practitioners, psychologists and other allied health professionals) have free access to the courses, while clients pay a small fee. Courses include a range of 'lessons' in a comic-based format that follows the progress of specific characters as they learn focused psychological skills, as well as downloadable resources, homework tasks, and recovery stories.
- The Inspire Foundation hosts *Reach Out.com* ([au.reachout.com](http://au.reachout.com)) which offers information, support and resources aimed at helping young people improve their understanding of mental health concerns, promote wellbeing and resilience, consider significant developmental issues such as sexuality and bullying, increase coping skills, and facilitate help seeking, help giving and networking.
- The Macquarie University Centre for Emotional Health runs *Mind Spot* ([www.mindspot.org.au](http://www.mindspot.org.au)), a free telephone and online service targeting stress, worry, anxiety, low mood and depression and providing mental health screening assessments, therapist-guided treatment and referrals.
- The Swinburne University of Technology National eTherapy Centre runs *Anxiety Online* and *Mental Health Online* (MHO; [www.mentalhealthonline.org.au](http://www.mentalhealthonline.org.au)), a comprehensive online mental health service offering information, assessment, online diagnosis, free access to treatment programs for clinicians, free self-help programs for clients and therapist-assisted treatment programs costing a small fee for clients. With an initial focus on anxiety disorders, MHO is expanding to include treatments for a broader range of mental health and health problems (e.g., psychosis, hoarding), multidagnostic programs, a virtual reality platform for individual and group therapy with the potential to use avatars to represent therapists and clients, and secure audio/videoconferencing facilities.

**Note:** While this article does not aim to offer a comprehensive review of all online supports, we also mention some additional initiatives as these are either unique (e.g., offering online resources for bipolar disorder — see Mood Swings [[www.moodswings.net.au](http://www.moodswings.net.au)]) or funded by State Government initiatives (e.g., On Track [[www.ontrack.org.au/web/ontrack/home](http://www.ontrack.org.au/web/ontrack/home)], a website run by the Queensland University of Technology offering free resources that aim to facilitate mental and physical health and wellbeing). The Beacon and mindhealthconnect portals can be used to source information about other online websites.

**Source:** Kyrios and Thomas (2014).

## The Third Wave of cognitive therapies

Subsequent variants of cognitive-behavioural therapies, particularly those collectively termed the Third Wave, have incorporated a varying focus on cognitive processes as distinct from content, self-schemas and related elements. Examples include acceptance and commitment therapy (ACT; Hayes, 2004; Hayes, Strosahl, & Wilson, 2016), **dialectical behaviour therapy** (Linehan, 1993), mindfulness-based cognitive therapy (Segal et al., 2013; Hayes, Follett, & Linehan, 2004), metacognitive therapy (Fisher & Wells, 2009) and post-rationalist approaches such as narrative therapy (Singer, 2004; White, 2007) and others (Guidano, 1991; Guidano & Liotti, 1983). However, the newer treatments differ from traditional CBT by incorporating a focus on spirituality, values, emotion, acceptance and mindfulness. According to Hayes (2004), the third-wave treatments characteristically can more easily integrate older, more traditional therapeutic approaches, are more adaptable to the assimilation of different therapeutic strategies and have a greater emphasis on a broader range of therapeutic changes and processes.

There has been some conjecture as to the efficacy of the Third Wave of therapies. Öst (2008) concluded that, despite showing moderate effectiveness, randomised controlled trials of third-wave therapies were methodologically less rigorous than those evaluating CBT and, subsequently, these therapies could not be considered to be empirically supported treatments. On the other hand, in a review of studies evaluating the efficacy of mindfulness-based therapies in treating anxiety and depression, Hofmann and colleagues (Hofmann, Sawyer, Witt, & Oh, 2010) concluded that they were moderately effective for both at post-treatment and at follow-up, with a better result for disorders than mood problems. Nonetheless, Hofmann, Sawyer, and Fang (2010) emphasised that, despite theoretical and procedural differences, third-wave therapies (specifically, ACT and mindfulness) were not really different to CBT as data does not confirm the efficacy of either over the other, nor does it support differences in mechanisms of action.

## Evaluating the cognitive–behavioural paradigm

The great advantage of the cognitive–behavioural approach is that it lends itself to scientific evaluation. As a result, it has found much support both as a theoretical framework that is easy to understand and as a mode of psychological treatment that is effective for a broad range of psychological disorders. However, much research is required to delineate the exact role of cognitive processes in psychopathology. There is more evidence to support that distorted thinking is a cause of psychological dysfunction and distress rather than a consequence of it. Nonetheless, much of the current research is focused on understanding what types of mechanisms sustain the biased thoughts shown in different psychopathologies, thus supporting more targeted treatments for specific disorders.

What is distinctive in the cognitive–behavioural paradigm is that the thoughts are given causal status; that is, the thoughts are regarded as causing the other features of the disorder, such as sadness. However, the cognitive therapy model has been criticised for its circular arguments in that it does not appear to explain much. That a person with depression has a negative schema tells us that the person thinks gloomy thoughts. But such a pattern of thinking is actually part of the diagnosis of depression. Left unanswered is the question of where the negative schema came from in the first place. Pertinent is the criticism that the cognitive approach is limited in the scope with which it considers developmental factors and emotional functioning. However, recent iterations of cognitive therapy which examine the developmental influences impacting on the emergence of specific beliefs and on self-constructs have overcome some of these criticisms. Of greater concern is the limited research examining sociocultural factors that impact on psychological functioning. Nonetheless, genetic and neuroscientific research also reflects limited attention to sociocultural influences on psychopathology, partly because such factors cut across the three main paradigms.

## 1.7 Factors that cut across the paradigms

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**LEARNING OUTCOME 1.7** Understand emotion, culture, ethnicity and interpersonal factors in the study and treatment of psychopathology.

Three important sets of factors that we will consider throughout this text are emotion, sociocultural and interpersonal factors. Such factors are important components of the *psychosocial* dimensions of the broader *biopsychosocial* framework of normal and abnormal behaviour that was introduced at the beginning of this chapter. Some type of disturbance in emotion can be found in nearly all psychological disorders. In addition, we will continuously see that gender, culture, ethnicity and social relationships bear importantly on the descriptions, causes and treatments of the different disorders. In the next sections, we introduce these concepts and give some examples of why they are so important in psychopathology, regardless of what paradigm has been adopted.

### Emotion and psychopathology

**Emotions** influence how we respond to problems and challenges in our environment; they help us organise our thoughts and actions, both explicitly and implicitly, and they guide our behaviour. Perhaps because our emotions exert such widespread influence, we spend a good deal of time trying to regulate how we feel and how we present our emotions to others. Given their centrality, it is not surprising that disturbances in emotion figure prominently in many different forms of psychopathology, although estimates vary. ‘It is commonly said that emotion dysregulation characterises more than half of the mental disorders described in the *Diagnostic and Statistical Manual of Mental Disorders*’ (Jazaieri, Urry, & Gross, 2013, p. 585). By another analysis, as many as 85 percent of psychological disorders include disturbances in emotional processing of some kind (Thoits, 1985).

What is emotion? The answer to that question could fill an entire text on its own. Emotions are believed to be fairly short-lived states, lasting for a few seconds, minutes or at most hours. Sometimes the word *affect* is used to describe short-lasting emotional feelings. Moods, on the other hand, are emotional experiences that endure for a longer period of time.

Most contemporary emotion theorists and researchers suggest that emotions have a number of components, including (but not limited to) expressive, experiential and physiological components. The *expressive*, or *behavioural*, *component* of emotion typically refers to facial expressions of emotion. Many people with schizophrenia display very few facial expressions. The *experience* or *subjective feeling*, *component* of emotion refers to how someone reports he or she feels at any given moment or in response to some event. For example, learning that you received an A on your mid-semester exam might elicit feelings of happiness, pride and relief. Learning you only received a pass might elicit feelings of anger, anxiety or embarrassment. The *physiological component* of emotion involves changes in the body, such as those due to the autonomic nervous system activity that accompanies emotion. For example, if a car almost runs you down as you are crossing the street, you may show a frightened look on your face, feel fear, and experience an increase in your heart rate and breathing rate.

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Emotion consists of many components, including expression (shown here), experience and physiology.



When we consider emotional disturbances in psychological disorders, it is important to consider which of the emotion components are affected. In some disorders, all emotion components may be disrupted, whereas in others, just one might be problematic. For example, people with schizophrenia do not readily express their emotions outwardly, but they report feeling emotions very strongly. People with panic disorder experience excessive fear and anxiety when no actual danger is present. People with germaphobia or OCD may feel disgust when confronted by what they think is a contaminated object. People with depression may experience prolonged sadness and other negative feelings. A person with

antisocial personality disorder may not feel empathy. We will try to be clear in the text as to what component of emotion is being considered.

## Sociocultural factors and psychopathology

A good deal of research has focused on the ways in which sociocultural factors, such as gender, race, culture, ethnicity and socioeconomic status, can contribute to different psychological disorders. Researchers who study such sociocultural factors and psychopathology all share the premise that environmental factors can trigger, exacerbate or maintain the symptoms that make up the different disorders. But the range of variables considered and the ways of studying those variables cover a lot of ground.

Several studies consider the role of gender in different disorders. These studies have shown that some disorders affect men and women differently. For example, depression is nearly twice as common among women as among men. On the other hand, antisocial personality disorder and alcohol use disorder are more common among men than women. Childhood disorders, such as attention-deficit hyperactivity disorder, affect boys more than girls, but some researchers question whether this reflects a true difference between boys and girls or a bias in the diagnostic criteria. Current research is looking beyond whether men and women differ in the prevalence rates of certain disorders to asking questions about **risk factors** that may differently impact men and women in the development of certain disorders. For example, father-to-son genetic transmission appears to be an important risk factor in the development of alcohol use disorder for men, whereas sociocultural standards of thinness may be a risk factor in the development of eating disorders for women.

Other studies show that poverty is a major influence on psychological disorders. For example, poverty is related to antisocial personality disorder, anxiety disorders and depression. We discuss the role of these factors in overall health in focus on discovery 1.4.

Cultural and ethnic factors in psychopathology have also been examined. Some questions relate to whether or not the disorders we diagnose and treat are observed across all parts of the world. Research has demonstrated that psychological disorder is observed across all parts of the world, but there are specific disorders that are more prevalent in some parts of the world and there are some disorders that present differently in some parts of the world relative to others.

In a later chapter, we consider the evidence that eating disorders are specific to Western culture. Murphy (1976) examined whether schizophrenia symptoms could be observed in cultures as diverse as the Eskimo and Yoruba. She found that both cultures have a concept of being ‘crazy’ that is quite similar to the Western definition of schizophrenia. The Eskimo’s *nuthkavihak* includes talking to oneself, refusing to talk, delusional beliefs and bizarre behaviour. The Yoruba’s *were* encompasses similar symptoms.

In Japan and Korea, one particularly common presentation of social anxiety disorder, *taijin kyofusho*, is characterised by a fear of interpersonal relations with a sense of extreme embarrassment or a fear of offending others via the functions of one’s body or appearance which is more common in males than females (James, 2006; Tarumi, Ichimiya, Yamada, Umesue, & Kuroki, 2004). The Japanese term *hikikomori* refers to a condition in which a person completely withdraws from his social world (although women are affected, it is observed predominantly among men). With respect to OCD, while cross-national prevalence rates are generally similar, there are differences in the importance of culturally-specific constructs and in the presentation of the disorder. For instance, affected people from the Middle East are more likely to present with concerns about purity and contamination relative to those from the West (Nedeljkovic, Moulding, Foroughi, Kyrios, & Doron, 2011). Current diagnostic systems do include cultural factors in the discussion of every category of disorder and this may be an important step towards increasing research in this area.

There is a range of factors that impact on the emergence, experience and efficacy of services for mental health conditions. A particularly important example of research is that of Indigenous populations. Focus on discovery 1.3 discusses how clashes of cultural and historical factors have impacted on the health and mental health of Aboriginal and Torres Strait Islander Australians.

### Health and mental health of Aboriginal and Torres Strait Islander Australians

In 2015, there were 729 048 Australians who identified as being from an Aboriginal or Torres Strait Islander background. A systematic review of findings from a range of publications and data sources, including national mental health surveys, reported prevalence rates for Indigenous people by age and sex and compared these with data from the general population (Jorm, Bourchier, Cvetkovski, & Stewart, 2012). Indigenous adults were consistently found to self-report higher rates of psychological distress. Overall, 26.6 percent of Indigenous adults self-reported mental health problems compared to 13.1 percent of non-Indigenous Australians in the 2004–05 National Health Survey, with generally similar findings and differences reported across other studies. Results from studies of Indigenous adolescents were more mixed, although surveys of parents and carers of Indigenous children and adolescents reported a higher prevalence of behaviour problems. For instance, the Western Australian Aboriginal Child Health Survey of children aged 4–17 years reported a prevalence rate of 24 percent for carer- or teacher-reported mental health problems in Indigenous children and adolescents compared to 15 percent for non-Indigenous counterparts. The authors concluded that inequality in mental health between Indigenous and non-Indigenous Australians starts from an early age. A more recent review (Australian Indigenous HealthInfoNet, 2016) reported that, relative to non-Indigenous people, Indigenous Australians were 2.7 times as likely as to feel high or very high levels of psychological distress. Hence, Indigenous Australians experience poorer health during their lives — there is a significant gap in their health and mental health relative to non-Indigenous Australians. Up to 10 percent of this gap has been linked to mental health conditions, with a further 4 percent accounted for by high **suicide** rates.

Alarming, Aboriginal and Torres Strait Islander peoples are 6 times more likely to commit suicide, compared to non-Indigenous people. The rate of intentional self-harm among Indigenous youth aged 15–24 years is over 5 times the rate of their non-Indigenous counterparts. The Kimberley region of Australia, with its high proportion of Indigenous people, has one of the highest suicide rates in the world, with particular ongoing increases among Indigenous youth and females over the last decade (Campbell, Chapman, McHugh, Sng, & Balaratnasingam, 2016).

Such statistics are reflected across a range of health and mental health areas related to Indigenous Australians. In 2013–14, there were over 16 000 hospitalisations with a principal diagnosis of mental and behavioural disorders in Aboriginal and Torres Strait Islander peoples with hospitalisation rates generally higher for Indigenous people than for other Australians, with notably higher rates for psychoactive substance use disorders, psychoses and intentional self-harm. With respect to health concerns, 13 percent of Aboriginal and Torres Strait Islander peoples had a long-term heart or related condition, around 1.2 times greater relative to non-Indigenous people. Similarly, 9 percent of Aboriginal and Torres Strait Islander peoples reported having diabetes, over 3 times the rate for non-Indigenous people, while the notification rate of end-stage renal disease was 6.6 times greater for Aboriginal and Torres Strait Islander peoples than for non-Indigenous people. While the rate of hospitalisation for injury was nearly twice the rate for Indigenous Australians relative to other Australians, alarmingly, the hospitalisation rate for assault was 34 times higher for Aboriginal and Torres Strait Islander women than for other Australian women. Despite such alarming statistics, there has been sustainable improvement in the gaps in Indigenous health and mental health outcomes, with particular improvements in infant mortality rates, life expectancy and mortality. It is generally agreed that the history of the treatment of Indigenous people globally has impacted on such health inequities.

Health inequities and differential treatment are related to the history of Indigenous-settler interactions, a misguided and failed ideology of tutelage, competition over resources located on Indigenous lands, power imbalances and cultural differences. (Lavoie et al., 2016, p. 1)

Similar health gaps can be found in the New Zealand population, with Maori and Pacific men and women 1.5 to 2 times more likely to have an anxiety or depressive disorder compared to the total New Zealand population (Ministry of Health, 2008) with racism a likely contributor (Harris, Cormack, & Stanley, 2013).

Culture-specific views of mental health, and social and emotional wellbeing among Indigenous people are holistic and focus on spirituality, wellness, harmony, balance, family and kin, community and the

individual's connections with their ancestors and traditional lands rather than symptoms and disorder (see Poroch et al., 2009). Culturally insensitive policies and services, and the disconnect between Indigenous cultural concepts and the traditional Western mental health framework, often undermine a sense of self-determination and are generally thought to have done little to compensate for the impact of the trauma of colonisation and disastrous social policies on Indigenous Australians (an example is the Stolen Generation, where, in an attempt to assimilate the Indigenous population into white society, Indigenous children were forcibly removed from their natural families).

The wellbeing of Indigenous populations cannot be fully understood without an appreciation of the events and processes that followed Australia's colonisation. Indigenous health is a product of a history of dispossession, exclusion, discrimination, marginalisation and inequality in various forms. Racism has affected a high proportion of Indigenous people in contemporary Australia. It has created a lack of trust between Indigenous and non-Indigenous people and impeded the process of healing and reconciliation. (Dudgeon et al., 2014, p. 6)

Culturally insensitive policies and services do little to compensate for disastrous social policies on Indigenous Australians. An example is the Stolen Generation.



While there has been discussion about the culturally inappropriate manner in which health and mental health services have been delivered to Indigenous communities, it is hoped that more collaborative initiatives will support better outcomes. Nonetheless, the current evidence base is meagre. A recent systematic review comparing the effectiveness of culturally adapted versus non-adapted interventions for Indigenous adults with mental or substance use disorders found 16 studies only across 4 countries (Australia, Canada, New Zealand and the United States) (see Leske et al., 2016). Relative to North American studies that had virtually no culturally adapted interventions, two-thirds of those from Australia and New Zealand had used culturally adapted interventions and included samples with mental disorders. Of the culturally non-adapted intervention studies, all but one reported significant improvements on at least one mental health measure. Of the culturally adapted intervention studies, all reported significant improvement on at least one mental health measure. The authors concluded that the evidence base regarding culture-based interventions remains inconclusive due to the small number of studies and their methodologically weaknesses. Hence, much work is still needed to develop effective interventions for culturally diverse populations.

Interestingly, there has also been widespread criticism of the way that the health and mental health professions have responded through the years to the challenges faced by Indigenous communities. The Australian Psychological Society was the first professional body to offer an apology to Australian Aboriginal and Torres Strait Islander peoples for the ways in which psychologists had failed to respond to the needs of Indigenous communities. The Apology called for new systems to improve engagement between the profession and Indigenous culture. The Apology can be viewed on the APS website: [www.psychology.org.au/news/media\\_releases/15September2016](http://www.psychology.org.au/news/media_releases/15September2016)

In advocating for the Apology, the APS considered it to be an important step in redressing past oversights and ensuring the psychology profession collaborated and appropriately served Aboriginal and Torres Strait Islander peoples into the future; but it also saw it as an important step in meeting the social and emotional wellbeing and mental health needs of people from an Indigenous background. Further information on how we can work together with Indigenous communities and psychologists to eliminate these gaps in their health and mental health relative to non-Indigenous Australians can be found in the publication *Working Together: Aboriginal and Torres Strait Islander Mental Health and Wellbeing Principles and Practice* edited by Purdie, Dudgeon, and Walker (2010).

### QUESTIONS

1. How do you think the history of colonisation and government policies such as those surrounding the Stolen Generation have impacted on Indigenous communities?
2. What are some strategies that might facilitate more culturally appropriate services for Indigenous people?

Sociocultural factors have also become more prominent in recent years in genetics and neuroscience. State and Geschwind (2015) note the importance of environmental influences:

There is now little argument that most pathology seen in the psychiatry clinic reflects complex genetics, involving extreme polygenicity, variable penetrance, tremendous heterogeneity and an important role for the environment. (p. 4)

In fact, cultural neuroscience is a fast-developing discipline that examines cultural variation in complex psychological, neural and genomic processes, providing new ways of studying the multifaceted interactions of these processes and their impact on individuals and populations (see Chiao et al., 2010). A multinational project called the 1000 Genomes Project ([www.1000genomes.org](http://www.1000genomes.org)) seeks to sequence the genomes of a representative sample of people around the world. In one study from this project, researchers found that people from each country had about the same number of rare variants in the genomes (Gravel et al., 2011). However, the rare variants were not the same in the different countries, suggesting that culture may also be influencing gene expression. For example, the short-short allele combinations of the 5-HTT gene studied by Caspi et al. (2003) varies across cultures (Chiao & Blizinsky, 2010, 2013). Cognitive-behavioural traditions have tended to focus more on the individual rather than on how the individual interacts with the social world. However, this, too, is changing. Efforts are underway, for example, to develop cognitive-behavioural therapy for people from different cultures and ethnicities (Bennett-Levy et al., 2014; Bennett & Babbage, 2014).

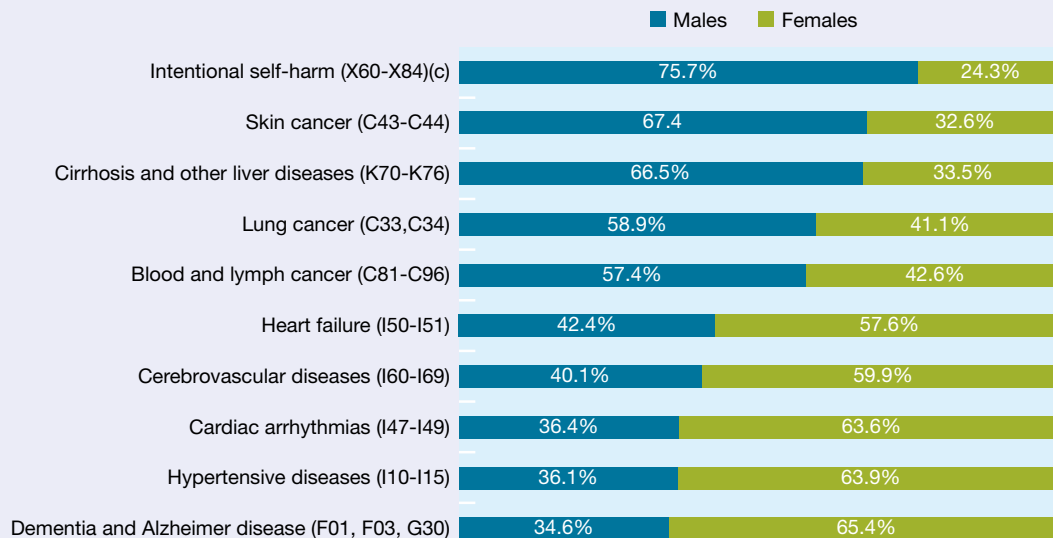
### FOCUS ON DISCOVERY 1.4

#### Sociocultural factors and health

Sociocultural factors such as gender, race, ethnicity and socioeconomic status are also important for understanding overall health. The many demonstrations of the pervasive role of these types of factors in health form the basis for the fields of **behavioural medicine** and **health psychology**. Here we consider how sociocultural factors have been studied in health psychology.

#### Gender and health

At every age from birth to 85 and older, more men die than women. Among the top 20 leading causes of death in Australia in 2015 (excluding sex-specific causes such as prostate and breast cancer), one sees that intentional self-harm and skin cancer were most likely to affect males, while hypertension and dementia, including Alzheimer's disease, predominantly affected females.

**FIGURE 1.19** Greatest sex ratios within the 20 leading causes of death, 2015

Source: ABS (2015).

What are some of the possible reasons for the differences in mortality and morbidity rates in men and women? It might be that women have some biological mechanism that protects them from certain life-threatening diseases. For example, epidemiological and observational studies suggested that estrogen might offer protection from cardiovascular disease. However, randomised clinical trials of hormone replacement therapy (HRT) for postmenopausal women (Hulley et al., 1998; Writing Group for the Women's Health Initiative Investigators, 2002) failed to find any such protective effects. In fact, a large prospective study had to end earlier than planned because women receiving estrogen plus progesterone therapy (so-called combined therapy) actually had an *increased* risk of breast cancer. Follow-up studies reveal that the risk of HRT varies quite a bit depending on a number of factors, such as a woman's age and other health risk factors. The most recent follow-up study suggests that in general, neither estrogen alone nor the combined therapy is helpful in reducing disease risk, but that small subgroups of women may benefit from particular types and timings (e.g., for a few years postmenopause) of HRT (Manson et al., 2013).

Other research has examined psychological risk factors for cardiovascular disease, such as anger and hostility. The evidence suggests that anger is not necessarily more commonly experienced and expressed by men, contrary to stereotypes about gender and emotion (Kring, 2000; Lavoie, Miller, Conway, & Fleet, 2001). However, increased hostility and both the suppression and expression of anger are associated with risk factors for cardiovascular disease among women (Matthews, Owens, Kuller, Sutton-Tyrrell, & Jansen-McWilliams, 1998; Rutledge et al., 2001). In addition, anxiety and depression are more common among women than men (see coming chapters) and are also linked to cardiovascular disease (e.g., Suls & Bunde, 2005).

Although the mortality rate is higher for men than women, the gender difference is shrinking. Why? In the early twentieth century, most deaths were due to infectious diseases, but now most deaths result from diseases that are affected by lifestyle. One possibility, then, is that lifestyle differences between men and women account for the sex difference in mortality and that these lifestyle differences are decreasing. Although men generally smoked more than women and consumed more alcohol, women are catching up in their use of alcohol and cigarettes, particularly younger women. Not surprisingly, then, these behaviour changes in women are paralleled by changing health profiles. Why do women have poorer health in general than men? There are several possible explanations, including their longevity, which makes them prone to certain diseases that are associated with ageing, like

Alzheimer's disease (see the chapter on late life and neurocognitive disorders). Women may be more attentive to their health than are men and thus may be more likely to visit physicians and be diagnosed. Women are exposed to more stress than men and they rate stress as having a greater impact on them, particularly stress related to major life events (Davis, Matthews, & Twamley, 1999). There is some evidence that medical practitioners treat women's health concerns less seriously than men's (Weisman & Teitelbaum, 1985). Finally, evidence indicates that women's morbidity differs depending on socioeconomic and demographic variables, such as income, education and ethnicity. For example, having more education and having a higher income are associated with fewer risk factors for cardiovascular disease, including obesity, smoking, hypertension and reduced amounts of exercise.

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Research shows that women live longer than men but women have more health problems than men.



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### QUESTIONS

1. What recent social changes do you think are impacting on the health of men and women?
2. What behavioural factors are associated with social changes that impact on the health of men and women?

## Interpersonal factors and psychopathology

Beyond the role of culture, ethnicity and poverty, thousands of articles have been published on how the quality of relationships influences different disorders. Family and marital relationships, social support and even the amount of casual social contact all play a role in influencing the course of disorders. In focus on discovery 1.5, we discuss interpersonal approaches to psychotherapy. Within relationships, researchers have looked for ways to measure not only the relative closeness and support offered but also the degree of hostility. The influence of stress within the context of social relationships plays a role in just about all the disorders we will consider. Other researchers are interested in understanding the role of trauma, serious life events and stress in psychopathology. In particular, many researchers have examined how the quality of our attachments to people, considered to be hugely influenced by the quality of our relationships with our parents, can affect the later development of specific disorders.

## Couples, family and interpersonal therapies

Given that interpersonal factors are important in nearly all disorders we discuss in this text, it is not surprising that therapies have been developed to focus on relationships.

### Couples therapy

Social and cultural changes have impacted on both marital and divorce patterns. While the marriage rate has been slowly declining over the past few decades, with the total number of divorces also generally declining more recently, one-third of marriages still end in divorce. Australian Bureau of Statistics figures show that, although the number of divorces increased by 4.3 percent in 2015, the overall divorce rate did not change from 2014 at 2.0 divorces per 1000 people. The average length of marriages ending in divorce was 12.1 in 2015, a slight increase from 12.0 years reported in 2014. In 2015, the median age at divorce for men was 45.3 years and 42.7 years for women.

While conflict is an inevitable part of any long-term partner relationship, people in a distressed marriage are two to three times as likely to experience a psychological disorder (Whisman & Uebelacker, 2006). In some couples, distress may be a consequence of the psychological disorder, but it is also clear that distress can contribute to psychological disorders. There are various strategies used to resolve severe relationship conflict, including working with a therapist when the relationship is in distress and taking a structured educational program (e.g., relationship education [RE]) to prevent the development of relationship issues.

In couples therapy, the therapist works with both partners together to reduce relationship distress. Treatments for most couples focus on improving communication, problem solving, satisfaction, trust and positive feelings. Halford, Pepping, and Petch (2015) report the large magnitude efficacy of couples therapy in randomised controlled trials, although they note that published studies examining its implementation in routine practice are associated with smaller effects. Furthermore, Halford and Bodenmann (2013) outline the usefulness of RE for couples who are currently satisfied in and committed to their relationship. Through a structured educational and skill-based approach, RE aims to promote a healthy relationship and prevent future conflict. There is excellent evidence that 9–20 hours of structured RE produces short- and longer-term improvements in couple satisfaction, particularly for those couples with modifiable risk factors (e.g., negative communication styles, poor problem solving, low self-regulation and poor dyadic coping).

### Family therapy

Family therapy is based on the idea that the problems of the family influence each member and that the problems of each member influence the family. As such, family therapy is used to address specific symptoms of a given family member, particularly for the treatment of childhood problems.

Some family therapists focus on roles within the family, asking questions about whether parents assume an appropriate level of responsibility. Sometimes family therapists attend to whether a given person in the family has been 'scapegoated', or unfairly blamed for a broader issue in the family. Many family therapists teach strategies to help families communicate and problem-solve more effectively.

Family therapy is often tailored to the specific disorder. In family approaches for conduct disorder, the therapist may focus on improving parental monitoring and discipline. For adolescents with other externalising problems, the goal of family therapy may be to improve communication, to change roles or to address a range of family problems. With disorders like schizophrenia and bipolar disorder, family therapy often includes psychoeducation as a supplement for the medication treatment provided to the individual. Psychoeducation focuses on improving understanding of the disorder, reducing expressed family criticism and hostility, and helping families learn skills for managing symptoms (Miklowitz, George, Richards, Simoneau, & Suddath, 2003). In anorexia nervosa, family members are used strategically to help the adolescent gain weight. In sum, the goals and strategies of family therapy will be adjusted to meet the needs of different clients. In childhood obsessive-compulsive disorder, engaging parents and family members in the treatment of affected individuals can decrease the family's accommodation to the disorder and lead to better outcomes, irrespective of whether the treatment is implemented in individual or group format (Barrett, Healy-Farrell, & March, 2004; Waters & Barrett, 2000).

### Interpersonal therapy

**Interpersonal therapy (IPT)** emphasises the importance of current relationships in a person's life and how problems in these relationships can contribute to psychological symptoms. The therapist first encourages the patient to identify feelings about his or her relationships and to express these feelings

and then helps the patient generate solutions to interpersonal problems. IPT has been shown to be an effective treatment for depression (discussed in more detail in the chapter on mood disorders). IPT has also been used to treat eating disorders, anxiety disorders and personality disorders.

In IPT, four interpersonal issues are assessed to examine whether one or more of them might be impacting symptoms:

- *unresolved grief* — for example, experiencing delayed or incomplete grieving following a loss
- *role transitions* — for example, transitioning from child to parent or from worker to retired person
- *role disputes* — for example, resolving different relationship expectations between romantic partners
- *interpersonal or social deficits* — for example, not being able to begin a conversation with an unfamiliar person or finding it difficult to negotiate with a boss at work.

In sum, the therapist helps the patient understand that psychopathology occurs in a social or relationship context and that getting a better handle on relationship patterns is necessary to reduce symptoms of psychopathology.

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Unresolved grief is one of the issues discussed in interpersonal therapy.



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### QUESTIONS

1. What are some of the potential advantages of preventative programs for relationship problems?
2. What are some of the advantages of including family members and examining a person's relationships in the treatment of psychological disorders?

## CLINICAL CASE

### Amina

Amina, a 17-year-old girl who lived with her parents and her 15-year-old brother, was referred for family-focused therapy (FFT) for bipolar disorder as an adjunct to medication treatment. She had received a diagnosis of bipolar I disorder in early adolescence and was treated with lithium carbonate and quetiapine but had never fully responded to medications.

During an individual assessment session, Amina explained that she thought about suicide almost daily and had made two prior attempts, both by overdosing on her parents' medications. Amina had kept both attempts secret from her parents. Ethically, clinicians need to take steps to keep a client safe, and in this case, one measure would be to let Amina's parents know about her suicidality. The clinician explained this to Amina.

The first goal in FFT is to provide psychoeducation about bipolar disorder. As the symptoms of bipolar disorder were being reviewed, the clinician asked Amina to discuss her suicide attempts with her parents. When Amina did so, her parents were surprised. Her father, who had experienced his own father's suicide, was particularly concerned.

After psychoeducation, a goal in FFT is to choose one problem for the family to address and to help them learn new problem-solving skills in the process. In this family, the focus of problem solving was how to keep Amina safe from her suicidal impulses. To begin problem solving, the therapist worked with the family to define the problem and its context. The therapist asked the family to discuss situations that seemed to place Amina at most risk for suicide. The family was able to pinpoint that both previous attempts had followed interpersonal losses.

The next phase of problem solving is to generate potential solutions. To help with this process, the clinician framed questions in the problem-solving process for the family, including whether Amina could share her suicidal thoughts with her parents, how to establish whether she was safe, what responses would be helpful from them and what other protective actions should be taken. Using this structure, the family was able to agree on the plan that Amina would phone or page her parents when she was feeling self-destructive. Amina and her parents generated a plan in which her parents would help Amina engage in positive and calming activities until her suicidal thoughts were less intrusive. Amina and her parents reported feeling closer and more optimistic.

The therapist then began to conduct the next phase of therapy, which focused directly on symptom management. This phase consisted of training Amina to monitor her moods, to identify triggers for mood changes and to help her cope with those triggers.

As is typical in FFT, the clinician introduced the communication enhancement module during session eight. A goal of this module is to role-play new communication skills. Family members practise skills such as 'active listening' by paraphrasing and labelling the others' statements and by asking clarifying questions. At first, Amina and her brother protested against the role-play exercises.

Amina experienced another loss during this period when her one and only close long-term friend announced that she was going to be moving out of state. Amina took an overdose of paracetamol in a suicide attempt. Soon after overdosing, she became afraid, induced vomiting and later told her parents about the attempt.

The next session focused on the suicide attempt. Her parents, particularly her father, were hurt and angry. Amina in turn reacted angrily and defensively. The therapist asked the family to practise active listening skills regarding Amina's suicidality. Amina explained that she had acted without even thinking about the family agreement because she had been so distressed about the idea of losing her friend. Amina's parents were able to validate her feelings using active listening skills. The therapist reminded the parents that suicidal actions are common in bipolar disorder and noted that Amina's ability to be honest about her suicide attempt was an indicator of better family connectedness. The therapist also recommended that Amina see her psychiatrist, who increased her dosage of lithium.

By the end of treatment after nine months, Amina had not made any more suicide attempts, had become more willing to take her medications and felt closer to her parents. Like many people with bipolar disorder, though, she remained mildly depressed. Amina and her family continued to see the therapist once every three months for ongoing support.

*Source:* Adapted from Miklowitz and Taylor (2005).



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### QUESTIONS

1. What are some of the useful personal skills that Amina was able to develop during the therapy sessions?
2. How do you think she will be able to use these in the future to help her cope?

## Attachment and the concept of the self in psychopathology

One of the central features of psychoanalysis is **transference**, which refers to a person's responses to the analyst that seem to reflect attitudes and ways of behaving towards important people in the patient's past, rather than reflecting actual aspects of the relationship between the person and the analyst. Contemporary psychodynamic theorists have built on the concept of transference to emphasise the importance of a person's interpersonal relationships for psychological wellbeing. One example is **object relations theory**, which stresses the importance of longstanding patterns in close relationships, particularly within the family, that are shaped by the ways in which people think and feel. The 'object' refers to another person in most versions of this theory. This theory goes beyond transference to emphasise the way in which a person comes to understand, whether consciously or not, how the self is situated in relation to other people. For example, a woman may come to understand herself as a worthless person based on her cold and critical relationship with her mother.

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Children who are securely attached to parents are more likely to be psychologically healthy adults.



Another influential theory, **attachment theory**, grew out of object relations theory. John Bowlby (1907–1990) first proposed this theory in 1969 and Mary Ainsworth (1913–1999) and colleagues (1978) developed a method to measure attachment styles in infants. The essence of the theory is that

the type or style of an infant's attachment to his or her caregivers can set the stage for psychological health or problems later in life. For example, infants who are securely attached to their caregivers are more likely to grow up to be psychologically healthy adults, whereas infants who are anxiously attached to their caregivers are more likely to experience psychological difficulties. Attachment theory has been extended to adults (Main, Kaplan, & Cassidy, 1985; Pietromonaco & Barrett, 1997) and couples (e.g., Fraley & Shaver, 2000); and therapies based on attachment theory have been developed for children and adults.

Social psychologists have integrated both of these theories into the concept of the *relational self*, which refers to the self in relation to others and is based on the experienced quality of one's personal attachments such as parent–child and romantic relationships, and relationships with significant others such as teachers and friends (Chen, Boucher, Andersen, & Saribay, 2013; Chen, Boucher, & Parker Tapias, 2006). The concept of the relational self has garnered a tremendous amount of empirical support. For example, people will describe themselves differently depending on what other close relationships they have been asked to think about (Chen et al., 2006). Other studies show that describing a stranger in terms that are similar to a description of a close significant other will trigger positive feelings and facial expressions, presumably linked to the view of the self in relation to the close other person (Andersen, Reznik, & Manzella, 1996). Thus, if you are given a description of a stranger you must interact with that resembles a description of a close friend from high school, you will be more likely to smile, perhaps as a result of thinking about yourself and your interactions with your high school friend.

Sedikides and Brewer (2001) distinguish three distinct aspects of the concept of self, explicating that individuals pursue a sense of personal identity by developing or highlighting unique characteristics (e.g., 'I am intelligent'), relationships with other individuals ('I am a good husband') and through their group memberships ('I am a psychology undergraduate student and we're different to all the other undergraduates'). Of particular note, John Turner and colleagues (Turner, 1985; Turner, Hogg, Oakes, Reicher, & Wetherell, 1987) conceptualised self-categorisation theory, which emerged from social identity theory. While social identity theory emphasises that intergroup discrimination is associated with the need to secure a positive social identity ('I am a likeable psychology student'), self-categorisation theory highlights that group behaviour can be described in terms of the social categorical self (e.g., ingroup versus outgroup, 'us' versus 'them', 'us women' versus 'us men', 'likeable psychology students like me' versus 'overly confident medical students'). Notably, social categorisation theory postulates that there are countless constructs and both conscious and non-conscious cognitive processes that individuals use to categorise and distinguish their social group and, therefore, themselves. At its very heart, self-categorisation is essentially 'comparative, inherently variable, fluid and context dependent' (Turner, Oakes, Haslam, & McGarty, 1994). Such constructs have important implications for social cohesion as categories can impact on how we perceive others within a social context. A recent Danish commercial relating to discrimination illustrates this beautifully — see [www.youtube.com/watch?v=jD8tjhVO1Tc](http://www.youtube.com/watch?v=jD8tjhVO1Tc).

More recently, the relevance of self-constructs has been reviewed from the perspective of understanding and treating psychological disorders (see Kyrios et al., 2016). Reviewing research examining self-constructs for the main DSM-5 disorder categories, Kyrios (2016) concluded that:

... the concept of the 'self' opens up many opportunities for advancing our understanding of human psychological functioning and dysfunction. It is also apparent that the use of this concept gives us multiple options to facilitate developments in the treatment of disorder and, perhaps, could improve the **efficacy** and **effectiveness** of therapy for a broad range of psychological conditions. (p. 233)

Furthermore, they review the way in which the self and identity are dealt with by major psychotherapies, including psychodynamic, CBT, and Third Wave and related approaches. Much work has yet to be done to empirically scrutinise the concept of self from a therapeutic perspective. However, it needs to be noted that, when self-constructs were incorporated into our understanding and the treatment of social anxiety, the magnitude of effects on outcomes increased (Gregory, Peters, & Rapee, 2016). Furthermore,

self-based constructs are not new to psychotherapy. Traditional counselling approaches, based on the **humanistic** movement in psychology, have long considered that humans are affected by their past, their environment and social relationships, as well as their motivations, which interact with their inherent abilities and self-determinism to set and work towards their goals. This philosophical view can be contrasted with the behavioural perspective, which is not concerned with internal processes, and from the psychoanalytic view, which assumes the importance of the unconscious in determining behaviour. Humanists do not agree with behaviourists and psychoanalysts, who reduce human nature to specific parts or processes rather than viewing them as complete wholes (Bugental, 1964). Carl Rogers was possibly the most influential of the humanists, although others such as Abraham Maslow, Otto Rank, Rollo May, Erich Fromm, Gordon Allport, Viktor Frankl, R. D. Laing, Fritz Perls, Thomas Szasz have played influential roles in highlighting individuals' inherent self-determinism, creativity and drive towards demonstrating and maximising their own capabilities, a process termed **self-actualisation**.

Carl Rogers is best known for his contributions to a therapeutic framework termed **person-centred therapy** (Rogers, 1953). Rogers saw three conditions as necessary for personal growth: **unconditional positive regard** (the idea that, irrespective, of the client's thoughts or behaviours, the therapist maintains a stance of acceptance and understanding), **empathy** (where the therapist listens and tries to understand the world from the client's perspective) and **congruence** (where the therapist maintains a genuineness and authenticity in their interactions with the client). While the CBT revolution in recent years has also maintained an approach of trying to understand the world from the client's perspective, there has been a tendency by some therapists to be reductionist and to treat the presenting disorder rather than understand the person (Kyrios, 2016). Nonetheless, person-centred therapy has been criticised for its lack of specificity and the lack of detailed therapeutic strategies to effect changes.

## 1.8 Integration across multiple levels of analysis: the diathesis–stress integrative paradigm

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**LEARNING OUTCOME 1.8** Recognise the importance of integration across multiple levels of analysis: the diathesis–stress integrative paradigm.

Psychopathology is much too diverse to be explained or treated adequately by any one of the current paradigms. Most of the disorders we will discuss in this text likely develop through an interaction of neurobiological and environmental factors, a view that we turn to next.

The **diathesis–stress** paradigm is an integrative paradigm that links genetic, neurobiological, psychological and environmental factors. It is not limited to one particular school of thought, such as cognitive–behavioural, genetic or neurobiological. The diathesis–stress concept was introduced in the 1970s as a way to account for the multiple causes of schizophrenia (Zubin & Spring, 1977). Its appeal continues today for many disorders, however, because, like the gene–environment interaction model reviewed above, it is a model that focuses on the interaction between a predisposition towards disease — the **diathesis** — and environmental, or life, disturbances — the stress. Diathesis refers most precisely to a constitutional predisposition towards illness, but the term may be extended to any characteristic or set of characteristics of a person that increases his or her chance of developing a disorder.

In the realm of neurobiology, for example, a number of disorders considered in later chapters appear to have a genetically transmitted diathesis. Other neurobiological diatheses include oxygen deprivation at birth, poor nutrition, and a maternal viral infection or smoking during pregnancy. Each of these conditions may lead to changes in the brain that predispose the individual towards psychopathology.

In the psychological realm, a diathesis for depression may be a particular cognitive schema or the chronic feeling of hopelessness sometimes found in people with depression. Other psychological diatheses include the ability to be easily hypnotised, which may be a diathesis for dissociative identity disorder (formerly called multiple personality disorder) and an intense fear of becoming fat, which is a vulnerability factor for some eating disorders.

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Stressors that may activate a diathesis range from minor, such as having car trouble, to major, such as the aftermath of bushfire.



Possessing the diathesis for a disorder increases a person's risk of developing it but does not by any means guarantee that a disorder will develop. The stress part of diathesis–stress is meant to account for how a diathesis may be translated into an actual disorder. In this context, stress generally refers to some noxious or unpleasant environmental stimulus that, in combination with the diathesis, triggers psychopathology. Psychological stressors include major traumatic events (e.g., becoming unemployed, divorce, death of a spouse, serving in combat) as well as more chronic stressors (e.g., living in poverty throughout childhood).

The key point of the diathesis–stress model is that both diathesis and stress are necessary in the development of disorders. Some people, for example, have inherited a predisposition that places them at high risk for mania (see the chapter on mood disorders): a certain amount of stress increases the possibility of developing mania. Other people, those at low genetic risk, are not likely to develop mania regardless of how difficult their lives are.

Another major feature of the diathesis–stress paradigm is that psychopathology is unlikely to result from the impact of any single factor. A genetically transmitted diathesis may be necessary for some disorders, but it is embedded in a network of other factors that also contribute to the disorder. These factors could include genetically transmitted diatheses for other personality characteristics; childhood experiences that shape personality; the development of behavioural competencies and coping strategies; stressors encountered in adulthood; cultural influences; and numerous other factors.

Finally, we should note that within this framework, the data gathered by researchers holding different paradigms are not incompatible with one another. For example, stress may be needed to activate a predisposition towards a problem in neurotransmitter systems. Some of the differences between the paradigms also appear to be more semantic than substantive. A cognitive–behavioural theorist may propose that maladaptive cognitions cause depression, whereas a neurobiological theorist may speak of the activity of a certain neural pathway. The two positions are not contradictory but merely reflect different levels of description, just as we could describe a table as pieces of wood in a particular configuration or as a collection of atoms.

In focus on discovery 1.6 we illustrate how adopting one paradigm to the exclusion of others can bias your view of the critical targets for treatment.

#### FOCUS ON DISCOVERY 1.6

##### Multiple perspectives on a clinical problem

To provide a concrete example of how it is possible to conceptualise a clinical case using multiple paradigms, we present a case and discuss how the information provided is open to a number of interpretations, depending on the paradigm adopted.

##### Arthur

Arthur's childhood had not been a particularly happy one. His mother died suddenly when he was only six and for the next 10 years he lived either with his father or with a maternal aunt. His father drank heavily, seldom managing to get through any day without some alcohol. His father's income was so irregular that he could seldom pay bills on time or afford to live in any but the most run-down neighbourhoods. At times Arthur's father was totally incapable of caring for himself, let alone his son. Arthur would then spend weeks, sometimes months, with his aunt in a nearby suburb.

Despite these early life circumstances, Arthur completed high school and entered university. He qualified for government financial assistance, but he also needed to work as a waiter and bartender to make ends meet. During these university years, he felt an acute self-consciousness with people he felt had authority over him — his boss, his lecturers and even some of his classmates, with whom he compared himself unfavourably.

Like many people at university, Arthur attended his fair share of parties, where he drank heavily. By his final year, however, he was drinking daily, often as a way to deal with the stress of studying and working at the same time.

Two years after university, Arthur married his girlfriend. He could never quite believe that his wife, as intelligent as she was beautiful, really cared for him. As the years wore on, his doubts about himself and about her feelings towards him would continue to grow. He felt she was far brighter than he and he worried that she would make more money than he would.

After university, Arthur began a job at a publishing company, serving as an editorial assistant. This job proved to be even more stressful than college. The deadlines and demands of the senior editors were difficult. He constantly questioned whether he had what it took to be an editor. Like his father, he often drank to deal with this stress.

Several years later, when it seemed that life should be getting easier, he found himself in even greater turmoil. Now 32 years old, with a fairly secure job that paid reasonably well, he was arguing more often with his wife. She continually complained about his drinking; he denied that there was a problem. After all, he was only drinking four beers a night. His wife wanted to start a family, but he was not sure if he wanted to have this additional stress in his life. His brooding over his marriage led him to drink even more heavily until finally, one day, he realised he was drinking too much and needed to seek help.

Depending on the paradigm you adopt, your conceptualisation of this case may differ. If you hold a genetic point of view, you are attentive to the family history, noting that Arthur's father had similar difficulties with alcohol. You are probably aware of the research (to be reviewed in the chapter on substance use disorders) that suggests a genetic contribution to alcohol use disorder. You do not discount environmental contributions to Arthur's problems and you hypothesise about the ways in which genetic factors interact with different environmental factors (e.g., stress and work and in his relationships), which may in turn increase the likelihood that he will turn to alcohol to cope.

Now suppose that you are committed to a cognitive-behavioural perspective, which encourages you to approach psychological disorders in terms of reinforcement patterns as well as cognitive variables. You may focus on Arthur's self-consciousness at university, which seems related to the fact that, compared with his fellow students, he grew up with few advantages. Economic insecurity and hardship may have made him unduly sensitive to criticism and rejection. Alcohol has been his escape from such tensions. But heavy drinking, coupled with persistent doubt about his own worth as a human being, has worsened an already deteriorating marital relationship, further undermining his confidence. As a cognitive-behavioural therapist, you may decide on cognitive-behavioural therapy to convince Arthur that he need not obtain universal approval for every undertaking.

If you adopt an integrative perspective, you might follow more than one of these strategies. You would acknowledge the likely genetic contribution to Arthur's alcohol use disorder, but you would also identify key triggers (e.g., job stress) that might lead to greater bouts of drinking. You would likely employ many of the therapeutic techniques noted in this chapter.

## QUESTIONS

1. What are the disadvantages of a purely simplistic explanation of Arthur's problems that focuses on only one paradigm?
2. Which of the paradigms would be most useful in helping Arthur feel confident in overcoming his problems?

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## SUMMARY

### 1.1 Describe the basic features of psychological disorder.

The basis that humans have used for understanding psychological disorder has varied through the ages, from supernatural to scientific. Modern definitions of psychological disorder contain several features with no single feature sufficient to differentiate normality from abnormality. Clinicians are interested in the impact on the life of affected individuals of their behaviours, thoughts and emotions. We also examine the degree of personal distress and disability or impairment in some important area of their life. Other important factors include the degree of threat or danger posed by the affected person's behaviour, as well as the frequency of those behaviours and whether social norms or expectations are challenged. Medical models assume biological differences, such as physiological features and genetic abnormalities, are the cause of psychopathology. Psychosocial paradigms highlight the importance of one's environment and experiences in shaping behaviour.

### 1.2 Understand stigma associated with psychological disorders and its historical context.

As a result of historical accounts about the aetiology of mental illness, affected people have been subjected to stigma and maltreatment. Early concepts of psychological disorders attributed odd behaviour to possession by evil spirits and led to unscientific and often inhumane treatment of affected individuals. While ancient Greek physicians took a more scientific approach to understanding and treating mental illness, the Dark Ages brought back the belief that supernatural forces caused psychological disorder. Nonetheless, the mid-1700s saw more humane management of people with mental disorders in Europe and the United States, despite an overemphasis at times on improperly evaluated biological treatments and the dominance of specific views about the primary importance of genes on behaviour. This history has impacted on the stigma associated with psychological disorder. Stigma encompasses ignorance or limitations in *knowledge* about mental health, which results in prejudice or negative *attitudes* to mental illness and discriminatory responses to people with mental health problems. Stigma can refer to the attitude of people towards those with mental health problems (*personal or public stigma*), internalised attitudes towards oneself that are unhelpful (*'self-stigma'*) and how one perceives the attitudes of others towards mental illness (*'perceived stigma'*). Stigma is experienced by some people or groups more than others and can vary for different cultures, religions, social and educational groups, ages and disorders. For instance, schizophrenia appears to be associated with more negative community attitudes than depression or anxiety. The consequences of stigma include discrimination, ostracism and greater social isolation, diminished opportunities for employment and social opportunities, shame, humiliation, despair and poorer help-seeking, making the burden of mental illness even greater.

### 1.3 Understand early psychological approaches to psychopathology.

From the eighteenth century on, various European practitioners took a more psychological approach to our understanding of psychological disorders, although the scientific foundations of such theories are now questionable. Nonetheless, from the work of the early mesmerists and hypnotists all the way through to the work of Freud and his psychoanalytic contemporaries, like Jung and Adler, greater emphasis was placed on the influence on behaviour of early developmental experiences and internal psychological structures and processes. The psychoanalysts not only emphasised the importance of early development, but also highlighted internal drives and unconscious conflicts. Such approaches led to the development of specific methods of treatment that to this day still influence how we manage psychological disorders.

### 1.4 Describe the essentials of the genetic paradigm.

The genetic paradigm focuses on questions such as whether certain disorders are heritable and, if so, what is actually inherited. Heritability is a population statistic, not a metric of the likelihood a particular person will inherit a disorder. Environmental effects can be classified as shared or

non-shared (sometimes called unique). Molecular genetics studies seek to identify differences in the sequence and structure of genes as well as gene polymorphisms, such as SNPs, that may be involved in psychopathology. Research has emphasised the importance of gene–environment interactions. In most cases, genes do their work via the environment. Recent examples of genetic influence being manifested only under certain environmental conditions (e.g., poverty and IQ; early maltreatment and depression) make clear that we must look not just for the genes associated with psychological disorders but also for the conditions under which these genes may be expressed.

### **1.5 Describe the essentials of the neuroscience paradigm.**

The neuroscience paradigm is concerned with the ways in which the brain contributes to psychopathology. Neurotransmitters such as serotonin, norepinephrine, dopamine and GABA have been implicated in a number of disorders. A number of different brain areas are also a focus of research. The autonomic nervous system, which includes the sympathetic and parasympathetic nervous systems, is also implicated in the manifestations of some disorders. The HPA axis is responsible for the body's response to stress and thus is relevant for several stress-related disorders. Stress can also produce problematic changes in the immune system and this can impact a number of psychological disorders, such as major depressive disorder. Cytokines help initiate such bodily responses to infection as fatigue, fever and activation of the HPA axis. Inflammation and higher levels of the cytokine IL-6 have been linked to a number of diseases in older adults. Biological treatments, primarily medications, are effective for different disorders, but they are not necessarily treating the cause of the problems, nor are they always the most effective treatments. Although the brain plays an important role in our understanding of the causes of psychopathology, we must be careful to avoid reductionism.

### **1.6 Describe the essentials of the cognitive–behavioural paradigm.**

The cognitive–behavioural paradigm reflects influences from behaviourism and cognitive science. Behaviour is thought to reflect conditioning, reinforcement schedules, modelling and other learning processes, as well as attention and cognition. From behaviourism, treatment techniques designed to alter the consequences or reinforcers of a behaviour, such as in time-out and exposure, are still used today. Cognitive science, on the other hand, focuses on concepts such as beliefs and schemas (a network of accumulated knowledge), attention, memory and the unconscious. These concepts form a major part of cognitive–behavioural theories and treatments of psychopathology. For example, research on implicit memory promoted acceptance of the ideas of unconscious influences on behaviour. Cognitive–behavioural therapy uses behaviour therapy techniques and cognitive restructuring. Aaron Beck's approach to cognitive–behavioural therapy has been particularly influential across a range of disorders. New cognitive and behavioural therapies have also emerged in recent years.

### **1.7 Understand emotion, culture, ethnicity and interpersonal factors in the study and treatment of psychopathology.**

Disturbances in emotion figure prominently in psychopathology, but the ways in which emotions can be disrupted vary quite a bit. Emotions guide our behaviour and help us to respond to problems or challenges in our environment. It is important to distinguish between components of emotion, including expression, experience and physiology. In addition, mood can be distinguished from emotion. Psychological disorders have different types of emotion disturbances and thus it is important to consider which of the emotion components are affected. In some disorders, all emotion components may be disrupted, whereas in others just one might be problematic. Emotion is an important focus in the paradigms.

Sociocultural factors, such as culture, ethnicity, gender and socioeconomic status, are important factors in the study of psychopathology. Some disorders, like schizophrenia or anxiety, appear to be universal across cultures, yet their manifestations may differ somewhat and the ways in which society regards them may also differ. Other disorders, like eating disorders or *taijin kyofusho*, may be specific to particular cultures. Some disorders are more frequently diagnosed in some ethnic groups than in others. The reasons for these differences are complex.

Sociocultural factors have recently become the focus of people working in the other paradigms and this trend will continue. Research has examined whether risk factors associated with various disorders differ for men and women. Interpersonal relationships have also been examined as important buffers against stress and have benefits for physical and mental health. Object relations theory, which has developed out of psychoanalytic concepts, stresses the importance of relationships, while its offshoot, attachment theory, which emphasises the role of attachment styles in infancy through adulthood, are also important in psychopathology research. Attachment-based concepts are important across all therapies and have been particularly emphasised by humanistic therapy.

### **1.8 Recognise the importance of integration across multiple levels of analysis: the diathesis–stress integrative paradigm.**

A ‘paradigm’ is a set of basic assumptions, which scientists use to understand the phenomena they are studying. Paradigms determine how to conceptualise and study a topic, what data to collect, how to interpret that data and how to collate different types of data. We examined three major contemporary paradigms, as well as some other frameworks, used in psychological research to understand normal and abnormal behaviour, cognition and emotions. The genetic, neuroscientific and cognitive–behavioural paradigms, as well as the psychodynamic and humanistic frameworks that were introduced, have helped us gather much data to better understand processes underpinning the development of psychological disorder and to develop strategies or approaches to treating disorders. We have also identified a number of factors that are important in all the various paradigms, including the impact of environment and experience, gender, culture and society, and the quality of our relationships. However, psychological disorder is complex and the lives of people are affected by all of the various factors that we have highlighted. Most of the disorders discussed in this text are influenced by the interaction of neurobiological and environmental factors. The diathesis–stress paradigm provides a framework for linking the importance of genetic, neurobiological, psychological and environmental factors in the emergence of psychopathology. Predispositions (i.e., the ‘diathesis’) constitute characteristics, which become particularly important in stressful or challenging situations, and it is this interaction that increases a person’s chance of developing a disorder but does not guarantee it. While some stressors are chronic and ongoing, others can be acute and overwhelming. We will see different models explaining how specific disorders develop, are maintained and are treated. Irrespective of the model one uses to understand disorder, the centrality of the *person* and our unconditional regard and respect for them must be maintained in the way they are treated.

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## **KEY TERMS**

**aetiology** all the factors that contribute to the development of an illness or disorder

**agonist** a drug that stimulates receptors normally specific to a particular neurotransmitter

**allele** any of the various forms of a particular gene

**amygdala** a subcortical structure of the temporal lobe involved in attention to emotionally salient stimuli and memory of emotionally relevant events

**analytical psychology** the psychoanalytical system of psychology developed and practised by Carl Gustav Jung

**anal stage** in psychoanalytic theory, the second psychosexual stage, which occurs during the second year of life when the anus is considered the principal erogenous zone

**animal model** the use in research of animals to study a disease, psychological or psychopathological process that is similar to a human condition

**antagonist** a drug that dampens the effect of a neurotransmitter on its receptors; for example, many dopamine antagonists block dopamine receptors

**anterior cingulate** in the subcortical region of the brain, the anterior portion of the cingulate gyrus, stretching about the corpus callosum

**anxiety** an unpleasant feeling of fear and apprehension accompanied by increased physiological arousal. Anxiety can be assessed by self-report, by measuring physiological arousal and by observing overt behaviour

**anxiety disorders** disorders in which fear or anxiety is overriding and the primary disturbance; including phobic disorders, social anxiety disorder, panic disorder, generalised anxiety disorder and agoraphobia

**archetype** a primitive mental image inherited from the earliest human ancestors and supposed to be present in the collective unconscious (from Jung's psychoanalytic theory)

**asylums** refuges established in Western Europe in the fifteenth century to confine and provide for the mentally ill; forerunners of the mental hospital

**attachment theory** the type or style of an infant's attachment to his or her caregivers can set the stage for psychological health or problems later in development

**attention-deficit hyperactivity disorder (ADHD)** a disorder in children marked by difficulties in focusing adaptively on the task at hand, inappropriate fidgeting and antisocial behaviour and excessive non-goal-directed behaviour

**autonomic nervous system (ANS)** the division of the nervous system that regulates involuntary functions; innervates endocrine glands, smooth muscle and heart muscle; and initiates the physiological changes that are part of the expression of emotion. See also sympathetic and parasympathetic nervous systems

**basal ganglia** part of the brain consisting of multiple subcortical nuclei situated at the base of the forebrain. Basal ganglia nuclei are strongly interconnected with the cerebral cortex, thalamus and brainstem, as well as several other brain areas. They are primarily responsible for motor control, as well as other roles such as motor learning, executive functions and behaviours and emotions

**behaviour genetics** the study of individual differences in behaviour that are attributable to differences in genetic make-up

**behaviour therapy** a branch of psychotherapy conceived narrowly as the application of classical and operant conditioning to the alteration of clinical problems but more broadly as applied experimental psychology in a clinical context

**behavioural activation (BA) therapy** clinical approach to depression that seeks to increase participation in positively reinforcing activities

**behavioural medicine** an interdisciplinary field concerned with integrating knowledge from medicine and behavioural science to understand health and illness and to prevent as well as treat psychophysiological disorders and other illnesses in which a person's psyche plays a role. See also health psychology

**behaviourism** the school of psychology originally associated with John B Watson, who proposed that observable behaviour, not consciousness, is the proper subject matter of psychology. Contemporary behaviourists do use mediational concepts, provided they are firmly anchored to observables

**benzodiazepines** any of several drugs commonly used to treat anxiety, such as Valium and Xanax

**biopsychosocial framework** a framework used to understand the importance of biological, psychological and social factors that impact on behaviour

**brainstem** the part of the brain connecting the spinal cord with the cerebrum; contains the pons and medulla oblongata, and functions as a neural relay station

**cardiovascular disease** medical problems involving the heart and the blood circulation system, such as hypertension or coronary heart disease

**catharsis** the psychoanalytic process of reliving an earlier emotional trauma and releasing emotional tension by expressing previously forgotten thoughts about a traumatic event

**caudate nucleus** a nucleus within the basal ganglia involved in learning and memory that is implicated in body dysmorphic disorder and obsessive-compulsive disorder

**cerebellum** an area of the hindbrain concerned with balance, posture and motor coordination

**cerebral cortex** the thin outer covering of each of the cerebral hemispheres; highly convoluted and composed of nerve cell bodies that constitute the grey matter of the brain

**cerebrum** the two-lobed structure extending from the brainstem and constituting the anterior (frontal) part of the brain; the largest and most recently developed portion of the brain, responsible for coordinating sensory and motor activities and performing higher cognitive processes

**chromosomes** the threadlike bodies within the nucleus of the cell, composed primarily of DNA and bearing the genetic information of the organism

**classical conditioning** a basic form of learning, sometimes referred to as Pavlovian conditioning, in which a neutral stimulus is repeatedly paired with another stimulus (called the unconditioned stimulus, UCS) that naturally elicits a certain desired response (called the unconditioned response, UCR). After repeated trials, the neutral stimulus becomes a conditioned stimulus (CS) and evokes the same or a similar response, now called the conditioned response (CR). Compare operant conditioning

**cognition** the process of knowing; the thinking, judging, reasoning and planning activities of the human mind. Behaviour is now often explained as depending on these processes

**cognitive-behavioural therapy (CBT)** behaviour therapy that incorporates theory and research on cognitive processes such as thoughts, perceptions, judgements, self-statements and tacit assumptions; a blend of both the cognitive and behavioural paradigms

**cognitive-behavioural paradigm** general view that people can best be understood by studying how they perceive and structure their experiences and how this influences behaviour

**cognitive biases** tendencies to perceive events in a negative manner, for example, by attending to or remembering negative information more than positive information; hypothesised to be driven by underlying negative schemas

**cognitive restructuring** any behaviour therapy procedure that attempts to alter the manner in which a client thinks about life so that he or she changes overt behaviour and emotions

**cognitive therapy** See cognitive restructuring. See also cognitive-behavioural therapy

**collective unconscious** Jung's concept that every human being carries within the wisdom, ideas and strivings of those who have come before

**conditioned response (CR)** See classical conditioning

**conditioned stimulus (CS)** See classical conditioning

**congruence** humanistic idea that the therapist must maintain a genuineness and authenticity in their interactions with the client

**conscience** a person's moral sense of right and wrong

**copy number variation (CNV)** refers to variation in gene structure involving copy number changes in a defined chromosomal region; could be in the form of a deletion where a copy is deleted or an addition (duplication) where an extra copy is added

**corpus callosum** the large band of nerve fibres connecting the two cerebral hemispheres

**cortisol** a 'stress hormone' secreted by the adrenal cortices; helps the body prepare to face threat

**cytokines** immune system molecules, released by activated macrophages, which help initiate such bodily responses to infection as fatigue, fever and activation of the HPA axis

**defence mechanism** in psychoanalytic theory, reality-distorting strategies unconsciously adopted to protect the ego from anxiety

**demonology** the doctrine that a person's abnormal behaviour is caused by an autonomous evil spirit

**depression** a mood state characterised by sadness and a lack of pleasure, particularly for events or situations in which one would usually experience pleasure

**diagnosis** the determination that the set of symptoms or problems of a patient indicates a particular disorder

**Diagnostic and Statistical Manual of Mental Disorders** See DSM-5

**dialectical behaviour therapy** a therapeutic approach to borderline personality disorder that combines client-centred empathy and acceptance with behavioural problem solving, social skills training and limit setting

**diathesis** predisposition towards a disease or abnormality

**diathesis–stress** as applied in psychopathology, a view that assumes that individuals predisposed towards a particular psychological disorder will be particularly affected by stress and will then manifest abnormal behaviour

**dopamine** central nervous system neurotransmitter, a catecholamine that is also a precursor of norepinephrine and apparently figures in schizophrenia and Parkinson’s disease

**DSM-5** the current *Diagnostic and Statistical Manual of Mental Disorders* of the American Psychiatric Association

**effectiveness** how well a therapeutic treatment works in the real world in the hands of broader samples of non-academic, less supervised therapists. Compare efficacy

**efficacy** how well a therapeutic treatment works under rarefied, academic research conditions. Compare effectiveness

**ego** in psychoanalytic theory, the predominantly conscious part of the personality, responsible for decision making and for dealing with reality

**emotion** the expression, experience and physiology that guide responses to problems and challenges in the environment

**empathy** the ability to understand and share the feelings of another; in therapy, it refers to the notion that the therapist listens and tries to understand the world from the client’s perspective

**enzyme** a complex protein that acts as a catalyst in regulating metabolic activities

**epigenetics** the study of changes in gene expression that are caused by something other than changes in the DNA (gene) sequence or structure, such as DNA methylation

**eugenics movement** a movement that aimed at improving the genetic composition of the human race through selective breeding and sterilisation

**exorcism** the casting out of evil spirits by ritualistic chanting or torture

**exposure** real-life (in vivo) or imaginal confrontation of a feared object or situation, especially as a component of systematic desensitisation. See also imaginal exposure

**extinction** the elimination of a classically conditioned response by the omission of the unconditioned stimulus; in operant conditioning, the elimination of the conditioned response by the omission of reinforcement

**extraversion** personality trait associated with frequent experiences of positive affect and social engagement

**fixation** in psychoanalytic theory, the arrest of psychosexual development at a particular stage through too much or too little gratification at that stage

**frontal lobe** the anterior portion of each cerebral hemisphere, in front of the central sulcus; active in reasoning and other higher mental processes

**gamma-aminobutyric acid (GABA)** inhibitory neurotransmitter that may be involved in the anxiety disorders

**gene** the smallest portion of DNA within a chromosome that functions as a piece of functional hereditary information

**gene expression** the switching on and off of the reading (transcription and translation) of genes into their products (usually proteins) and thus their associated phenotypes

**gene–environment interaction** the influence of genetics on an individual’s sensitivity or reaction to an environmental event

**genetic paradigm** the approach to human behaviour that focuses on both heritability of traits and complex interactions between genes and environment

**genital stage** in psychoanalytic theory, the final psychosexual stage, reached in adulthood, in which heterosexual interests predominate

**genome-wide association studies (GWAS)** studies of variations in the entire human genome to identify associations between variations in genes and particular behaviours, traits or disorders. Large sample sizes are needed for these types of studies

**genotype** an individual's unobservable, genetic constitution, that is, the totality of genes present in the cells of an individual; often applied to the genes contributing to a single trait. Compare phenotype

**grey matter** the neural tissue — made up largely of nerve cell bodies — that constitutes the cortex covering the cerebral hemisphere, the nuclei in lower brain areas, columns of the spinal cord and the ganglia of the autonomic nervous system. Compare white matter

**gyrus** a ridge or fold between two clefts on the cerebral surface in the brain

**habituation** the diminishing of an innate response to a frequently repeated stimulus

**harmful dysfunction** proposed definition of psychological disorder that contains both a value judgement (harmful) and a putatively objective scientific component (dysfunction)

**health psychology** a branch of psychology dealing with the role of psychological factors in health and illness. See also behavioural medicine

**heritability** the extent to which variability in a particular behaviour/disorder within a population can be attributed to genetic factors

**hippocampus** in the subcortical region of the brain, the long, tubelike structure that stretches from the septal area into the temporal lobe

**HPA axis** the neuroendocrine connections among hypothalamus, pituitary gland and adrenal cortex, central to the body's response to stress

**humanistic** referring to humanism, a psychological perspective that emphasises the study of the whole person and looks at human behaviour through the eyes of the observer and the individual person

**hypnosis** a trancelike state or behaviour resembling sleep, induced by suggestion, characterised primarily by increased suggestibility

**hypothalamus** in the subcortical region of the brain, the structure that regulates many visceral processes, including metabolism, temperature, perspiration, blood pressure, sleeping and appetite

**id** in psychoanalytic theory, that part of the personality present at birth, comprising all the energy of the psyche and expressed as biological urges that strive continually for gratification

**identity** the qualities, beliefs, personality and other characteristics that make a person (self-identity) or group (particular social category or social group) unique. The process of identity can be creative or destructive

**in vivo** as applied in psychology, taking place in a real-life situation

**individual psychology** body of theories by Alfred Adler, who held that the main motives of human thought and behaviour are individual man's striving for superiority and power

**inhibitory learning** learning which inhibits previous learning

**interpersonal therapy (IPT)** a short-term, here-and-now focused psychological treatment initially developed for depression and influenced by the psychodynamic emphasis on relationships

**introversion** personality trait associated with frequent focus on one's internal experiences; introverts are typically perceived as more reserved or reflective

**latency period** in psychoanalytic theory, the years between ages 6 and 12, during which id impulses play a minor role in motivation

**law of effect** a principle of learning that holds that behaviour is acquired by virtue of its consequences

**libido** Freudian term for the life-integrating instinct or force of the id; sometimes equated with sexual drive

**limbic system** a complex system of nerves and networks in the brain that is concerned with instinct and mood and controls basic emotions (fear, pleasure, anger) and drives (hunger, sex, dominance, nurturance)

**major depressive disorder (MDD)** a disorder of individuals who have experienced episodes of depression but not of mania. Depression episodes are marked by sadness or loss of pleasure, accompanied by symptoms such as feelings of worthlessness and guilt; withdrawal from others; loss of sleep, appetite or sexual desire; and either lethargy or agitation

**mesmerise** the first term for hypnotise, after Franz Anton Mesmer, an Austrian physician, who in the late eighteenth century treated and cured hysterical or conversion disorders with what he considered the animal magnetism emanating from his body and permeating the universe

**mindfulness-based cognitive therapy (MBCT)** recent adaptation of cognitive therapy/restructuring that focuses on relapse prevention after successful treatment for recurrent episodes of major depression; aims to 'decentre' the person's perspective in order to break the cycle between sadness and thinking patterns

**modelling** learning by observing and imitating the behaviour of others or teaching by demonstrating and providing opportunities for imitation

**molecular genetics** studies that seek to determine the components of a trait that are heritable by identifying relevant genes and their functions

**mood disorders** disorders, such as depressive disorders or mania, in which there are disabling disturbances in emotion

**moral treatment** a therapeutic regimen, introduced by Philippe Pinel during the French Revolution, whereby mentally ill patients were released from their restraints and were treated with compassion and dignity rather than with contempt and denigration

**negative reinforcement** the strengthening of a tendency to exhibit desired behaviour by rewarding responses in that situation with the removal of an aversive stimulus

**neuron** a single nerve cell

**neuroplasticity** the brain's capacity to change and adapt in response to learning or experience or following injury, particularly referring to the physiological changes and the ability to form and reorganise synaptic connections

**neuroscience paradigm** a broad theoretical view that holds that psychological disorders are caused in part by some aberrant process directed by the brain

**neurotransmitters** chemical substances important in transferring a nerve impulse from one neuron to another, for example, serotonin and norepinephrine

**non-shared environment** factors distinct among family members, such as relationships with friends or specific experiences unique to a person. Compare shared environment

**norepinephrine** a catecholamine neurotransmitter, disturbances of which have been related to mania, depression and particularly to anxiety disorders. It is also a sympathetic nervous system neurotransmitter, a hormone released in addition to epinephrine and similar in action and a strong vasoconstrictor

**object relations theory** variant of psychoanalytic theory that focuses on the way children internalise (introject) images of the people who are important to them (e.g., their parents), such that these internalised images (object representations) become part of the ego and influence how the person reacts to the world

**obsessive-compulsive disorder (OCD)** a disorder characterised by high levels of distress and anxiety in which the mind is flooded with persistent and uncontrollable thoughts or the individual is compelled to repeat certain acts again and again, causing significant interference with everyday functioning

**occipital lobe** the posterior portion of each cerebral hemisphere, situated behind the parietal lobe and above the temporal lobes; responsible for reception and analysis of visual information and for some visual memory

**operant conditioning** the acquisition or elimination of a response as a function of the environmental contingencies of reinforcement and punishment. Compare classical conditioning

**oral stage** in psychoanalytic theory, the first psychosexual stage, which extends into the second year; during this stage the mouth is the principal erogenous zone

**paradigm** a set of basic assumptions that outlines the universe of scientific inquiry, specifying both the concepts regarded as legitimate and the methods to be used in collecting and interpreting data

**parasympathetic nervous system** the division of the autonomic nervous system that is involved with maintenance; controls many of the internal organs and is active primarily when the organism is not aroused. Compare sympathetic nervous system

**parietal lobe** the middle division of each cerebral hemisphere, situated behind the central sulcus and above the lateral sulcus; the receiving centre for sensations of the skin and of bodily positions

**person-centred therapy** an approach to therapy and counselling, based on the work of Rogers, that places much of the responsibility for the treatment process on the client, with the therapist taking a non-directive role

**phallic stage** in psychoanalytic theory, the third psychosexual stage, extending from ages 3 to 5 or 6, during which maximal gratification is obtained from genital stimulation

**phenotype** the totality of physical characteristics and behavioural traits of an individual or a particular trait exhibited by an individual; the product of interactions between genetics and the environment over the course of development. Compare genotype

**phobia** an anxiety disorder in which there is intense fear and avoidance of specific objects and situations, recognised as irrational by the individual

**pleasure principle** in psychoanalytic theory, the demanding manner by which the id operates, seeking immediate gratification of its needs

**polygenic** as applied to psychopathology or any other trait, caused by multiple genes contributing their effects, typically during multiple stages of development

**polymorphism** any specific difference in DNA sequence that exists within a population

**positive reinforcement** the strengthening of a tendency to exhibit desired behaviour by rewarding responses in that situation with a desired reward

**post-traumatic stress disorder (PTSD)** an anxiety disorder in which a particularly stressful event, such as military combat, rape or a natural disaster, brings in its aftermath intrusive reexperiencing of the trauma, a numbing of responsiveness to the outside world, estrangement from others and a tendency to be easily startled, as well as nightmares, recurrent dreams and otherwise disturbed sleep

**prefrontal cortex** the region of the frontal lobe of the brain that helps maintain an image of threats and rewards faced, as well as maintain focus and plan relevant to those threats and rewards

**prevalence** in epidemiological studies of a disorder, the percentage of a population that has the disorder at a given time

**prognosis** a prediction of the likely course and outcome of an illness. Compare diagnosis

**pruning** in neural development, the selective loss of synaptic connections, especially in the fine-tuning of brain regions devoted to sensory processing

**psyche** in psychoanalytic theory, the totality of the id, ego and superego, including both conscious and unconscious components

**psychoactive** refers to prescribed chemical compounds — for example, Prozac — having a psychological effect that alters mood or thought process

**psychoanalysis** primarily the therapy procedures pioneered by Freud, entailing free association, dream analysis and working through the transference neurosis. More recently, the term has come to encompass the numerous variations on basic Freudian therapy

**psychoanalytic theory** theory originating with Freud that psychopathology results from unconscious conflicts in the individual

**psychological disorder** a clinically significant behavioural or psychological syndrome or patterns. The definition includes a number of key features, including distress, disability or impaired functioning, violation of social norms and dysfunction

**psychoneuroimmunology** field that studies how psychological factors (especially stressors) impact the immune system (adversely)

**psychopathology** the field concerned with the nature and development of psychological disorders

**psychotherapy** a primarily verbal means of helping troubled individuals change their thoughts, feelings and behaviour to reduce distress and to achieve greater life satisfaction

**publication bias** the tendency to publish only positive results in journals

**rational-emotive behaviour therapy (REBT)** a cognitive-behavioural therapy developed by Albert Ellis

**reality principle** in psychoanalytic theory, the manner in which the ego delays gratification and otherwise deals with the environment in a planned, rational fashion

**receptor** a protein embedded in a neural cell membrane that interacts with one or more neurotransmitters. Non-neural receptor proteins include hormone receptors

**reuptake** cellular process by which released neurotransmitters are taken back into the presynaptic cell, terminating their present postsynaptic effect but making them available for subsequent modulation of nerve impulse transmission

**risk factor** a condition or variable that increases the likelihood of developing a disorder

**schema** a mental structure for organising information about the world

**schizophrenia** a disorder characterised by disturbances in thought, emotion and behaviour; disordered thinking in which ideas are not logically related; delusional beliefs; faulty perception, such as hallucinations; disturbances in attention; disturbances in motor activity; blunted expression of emotion; reduced desire for interpersonal relations and withdrawal from people; diminished motivation and anticipatory pleasure

**self** how someone thinks about, evaluates or perceives themselves; what distinguishes a person from others

**self-actualisation** the realisation or fulfilment of one's potential, considered as a drive or need present in everyone

**self-schemas** schemas relating to the self (see schema)

**septal area** in the subcortical region of the brain, the area anterior to the thalamus

**serotonin** a neurotransmitter of the central nervous system whose disturbances apparently figure in depression

**serotonin transporter gene** a particular gene critical to the gene–environment interactions that apparently contribute to the development of depression

**shared environment** factors that family members have in common, such as income level, child-rearing practices and parental marital status and quality. Compare non-shared environment

**single nucleotide polymorphism (SNP)** a variation in gene sequence; specifically, differences between people in a single nucleotide (A, T, G or C) in the DNA sequence of a particular gene

**stigma** the pernicious beliefs and attitudes held by a society, ascribed to groups considered deviant in some manner, such as people with mental illness

**stimulant** a drug, such as cocaine, that increases alertness and motor activity and at the same time reduces fatigue, allowing an individual to remain awake for an extended period of time

**stress** state of an organism subjected to a stressor; can take the form of increased autonomic activity and in the long term can cause breakdown of an organ or development of a psychological disorder

**sulcus** a groove or furrow on the surface of the brain

**suicide** the intentional taking of one's own life

**superego** in psychoanalytic theory, the part of the personality that acts as the conscience and reflects society's moral standards as learned from parents and teachers

**sympathetic nervous system** the division of the autonomic nervous system that acts on bodily systems — for example, contracting the blood vessels, reducing activity of the intestines and increasing the heartbeat — to prepare the organism for exertion, emotional stress or extreme cold. Compare parasympathetic nervous system

**symptom** an observable physiological or psychological manifestation of a disease

**synapse** small gap between two neurons where the nerve signal passes electrically or chemically from the axon of the first to the dendrites, cell body or axon of the second

**systematic desensitisation** a major behaviour therapy procedure that has a fearful person, while deeply relaxed, imagine a series of progressively more fearsome situations, such that fear is dispelled as a response incompatible with relaxation; useful for treating psychological problems in which anxiety is the principal difficulty

**temporal lobe** a large region of each cerebral hemisphere situated below the lateral sulcus and in front of the occipital lobe; contains primary auditory and general association areas

**thalamus** a major brain relay station consisting of two egg-shaped lobes; receives impulses from all sensory areas except the olfactory and transmits them to the cerebrum for higher processing

**theory** a formally stated and coherent set of propositions that explain and logically order a range of phenomena, generating testable predictions or hypotheses

**time-out** an operant conditioning procedure in which, after bad behaviour, the person is temporarily removed from a setting where reinforcers can be obtained and placed in a less desirable setting, for example, in a boring room

**transcription** in genetics, the first step in gene expression. A section of DNA sequence is transcribed to RNA; a sequence of DNA synthesises a copy of RNA

**transference** the venting of the analysand's emotions, either positive or negative, by treating the psychoanalyst as the symbolic representative of someone important in the past

**unconditional positive regard** the idea that, irrespective of the client's thoughts or behaviours, the therapist maintains a stance of acceptance and understanding

**unconditioned response (UCR)** See classical conditioning

**unconditioned stimulus (UCS)** See classical conditioning

**unconscious** a state of unawareness without sensation or thought; in psychoanalytic theory, the part of the personality, in particular the id impulses or energy, of which the ego is unaware

**unconscious conflict** a psychoanalytic notion that conflicts within the person that are not part of conscious experience affect our lives and functioning. Freud called this 'psychic determinism'

**ventricles** cavities deep within the brain, filled with cerebrospinal fluid, that connect to the spinal cord

**white matter** neural tissue, particularly of the brain and spinal cord, consisting of tracts or bundles of myelinated (sheathed) nerve fibres. Compare grey matter

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## WEBSITES

1. Sponsored by the Australian government, this website aggregates mental health resources and content from the leading mental health organisations in Australia and provides access to trusted online mental health resources. ([www.mindhealthconnect.org.au](http://www.mindhealthconnect.org.au))
2. Sane Australia provides information about serious mental illness for families, carers and affected people. It includes inspiring stories from people affected by complex mental illness that show how social connection and support can make a meaningful difference. ([www.sane.org](http://www.sane.org))
3. Beyond Blue provides information and resources for people who want to know more about depression and anxiety. ([www.beyondblue.org.au](http://www.beyondblue.org.au))
4. Black Dog Institute provides information and resources about mental illness with a focus on depression and bipolar disorder. ([www.blackdoginstitute.org.au](http://www.blackdoginstitute.org.au))

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## REFERENCES

- Adler, A. (1930). *Guiding the child on the principles of individual psychology*. New York: Greenberg.
- Ainsworth, M. S., Blehar, M. C., Waters, E., & Wall, S. (1978). *Patterns of attachment: A psychological study of the strange situation*. Oxford, UK: Erlbaum.
- Allderidge, P. (1979). Hospitals, mad houses, and asylums: Cycles in the care of the insane. *British Journal of Psychiatry*, 134, 321–324.
- Amir, N., Foa, E. B., & Coles, M. E. (1998). Negative interpretation bias in social phobia. *Behaviour Research and Therapy*, 36, 945–957.

- Andersen, S. M., Reznik, I., & Manzella, L. M. (1996). Eliciting transient affect, motivation, and expectancies in transference: Significant-other representations and the self in social relations. *Journal of Personality and Social Psychology*, 71, 1108–1129.
- Atlantis, E., Sullivan, T., Sartorius, N., & Almeida, O. P. (2012). Changes in the prevalence of psychological distress and use of antidepressants or anti-anxiety medications associated with comorbid chronic diseases in the adult Australian population, 2001–2008. *Australian and New Zealand Journal of Psychiatry*, 46, 445–456.
- Australian Bureau of Statistics (ABS). (2011). *Patterns of use of mental health services and prescription medications, 2011*, cat. no. 4329.0.00.003. Retrieved from [www.abs.gov.au](http://www.abs.gov.au)
- Australian Bureau of Statistics (ABS). (2015). *Causes of death, Australia, 2015*, cat. no 3303.0. Retrieved from [www.abs.gov.au](http://www.abs.gov.au)
- Australian Indigenous HealthInfoNet. (2016). *Overview of Australian Indigenous and Torres Strait Islander health status, 2015*. Perth, WA: Australian Indigenous HealthInfoNet.
- Australian Psychological Society (APS). (2016, September 15). *Australian Psychological Society apologises to Aboriginal and Torres Strait Islander People* [Media release]. Retrieved from [www.psychology.org.au/news/media\\_releases/15September2016](http://www.psychology.org.au/news/media_releases/15September2016)
- Bakker, J. M., Lieveise, R., Menne-Lothmann, C., Viechtbauer, W., Pishva, E., Kenis, G., . . . Wichers, M. (2014). Therapy genetics in mindfulness-based cognitive therapy: Do genes have an impact on therapy-induced change in real-life positive affective experiences? *Translational Psychiatry*, 4, 1–10.
- Bandura, A., Blanchard, E. B., & Ritter, B. (1969). Relative efficacy of desensitization and modeling approaches for inducing behavioral, affective, and attitudinal changes. *Journal of Personality and Social Psychology*, 13, 173–199.
- Bandura, A., & Menlove, F. L. (1968). Factors determining vicarious extinction of avoidance behavior through symbolic modeling. *Journal of Personality and Social Psychology*, 8, 99–108.
- Barrett, P., Healy-Farrell, L., & March, J. S. (2004). Cognitive-behavioral family treatment of childhood Obsessive-Compulsive Disorder: A controlled trial. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 46–62. doi:10.1097/00004583-200401000-00014
- Baxter, L. R., Ackermann, R. F., Swerdlow, N. R., Brody, A., Saxena, S., Schwartz, J. M., . . . Phelps, M. E. (2000). Specific brain system mediation of obsessive-compulsive disorder responsive to either medication or behavior therapy. In W. K. Goodman, M. V. Rudorfer & J. D. Maser (Eds.), *Obsessive-compulsive disorder: Contemporary issues in treatment* (pp. 573–610). Mahwah, NJ: Lawrence Erlbaum.
- Beck, A. T. (1967). *Depression: Clinical, experimental and theoretical aspects*. New York: Harper & Row.
- Beck, A. T. (2005). The current state of cognitive therapy: A 40-year retrospective. *Archives of General Psychiatry*, 62, 953–959.
- Bennett, S. T., & Babbage, D. R. (2014). Commentary: Cultural adaptation of CBT for Aboriginal Australians. *Australian Psychologist*, 49, 19–21. doi:10.1111/ap.12029
- Bennett-Levy, J., Wilson, S., Nelson, J., Stirling, J., Ryan, K., Rotumah, D., . . . Beale, D. (2014). Can CBT be effective for Aboriginal Australians? Perspectives of Aboriginal practitioners trained in CBT. *Australian Psychologist*, 49(1), 1–7. doi:10.1111/ap.12025
- Bockhoven, J. (1963). *Moral treatment in American psychiatry*. New York: Springer-Verlag.
- Brady, S., Britton, J., & Grover, S. (2001). Chapter two: The debate about numbers. In *The sterilisation of girls and young women in Australia: Issues and progress (2001)*. Australian Human Rights Commission. Retrieved from [www.humanrights.gov.au/publications/sterilisation-chapter-two](http://www.humanrights.gov.au/publications/sterilisation-chapter-two)
- Bransford, J. D., & Johnson, M. K. (1973). Considerations of some problems of comprehension. In W. G. Chase (Ed.), *Visual Information Processing*. New York: Academic Press.
- Brooks, M. (2004). *Extreme measures: The dark visions and bright ideas of Francis Galton*. London: Bloomsbury.
- Bugental, J. (1964). The third force in psychology. *Journal of Humanistic Psychology*, 4(1), 19–26. doi:10.1177/002216786400400102
- Bulik-Sullivan, B., Finucane, H. K., Anttila, V., Gusev, A., Day F. R., Loh, P. R. . . . Neale, B. M. (2015). An atlas of genetic correlations across human diseases and traits. *Nature Genetics*, 47, 1236–1244.
- Butler, A. C., Chapman, J. E., Foreman, E. M., & Beck, A. T. (2006). The empirical status of cognitive behavioral therapy: A review of meta-analyses. *Clinical Psychology Review*, 26, 17–31. doi: 10.1016/j.cpr.2005.07.003
- Campbell, A., Chapman, M., McHugh, C., Sng, A., & Balaratnasingam, S. (2016). Rising Indigenous suicide rates in Kimberley and implications for suicide prevention. *Australasian Psychiatry*, 24, 561–564. doi: 10.1177/1039856216665281
- Carr, E. G., Levin, L., McConnachie, G., Carlson, J. I., Kemp, D. C., & Smith, C. E. (1994). *Communication based intervention for problem behavior*. Baltimore: Paul H. Brookes.
- Carr, V., & Halpin, S. (2002). *Stigma and discrimination: A Bulletin of the Low Prevalence Disorders Study*. Canberra: Publications Production Unit (Governance and Business Strategy Branch), Commonwealth of Australia.
- Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry*, 167, 509–527.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301, 386–389.
- Chabris, C. F., Lee, J. J., Benjamin, D. J., Beauchamp, J. P., Glaeser, E. L., Borst, G., et al. (2013). Why it is hard to find genes associated with social science traits: Theoretical and empirical considerations. *American Journal of Public Health*, 103 Suppl 1, S152–166.
- Chase, A. (1980). *The legacy of Malthus*. Urbana: University of Illinois Press.
- Chen, S., Boucher, H. C., & Parker Tapias, M. (2006). The relational self revealed: Integrative conceptualization and implications for interpersonal life. *Psychological Bulletin*, 132, 151–179.

- Chen, S., Boucher, H. C., Andersen, S. M., & Saribay, S. A. (2013). *Transference and the relational self*. New York: Oxford University Press.
- Chiao, J. Y., & Blizinsky, K. D. (2010). Culture-gene coevolution of individualism-collectivism and the serotonin transporter gene. *Proceedings of the Royal Society B*, 277, 529–537.
- Chiao, J. Y., & Blizinsky, K. D. (2013). Population disparities in mental health: Insights from cultural neuroscience. *American Journal of Public Health*, 103(S1), S122–S132.
- Chiao, J. Y., Hariri, A. R., Harada, T., Mano, Y., Sadato, N., Parrish, T. B., & Iidaka, T. (2010). Theory and methods in cultural neuroscience. *Social Cognitive Affective Neuroscience*, 5, 356–361. doi:10.1093/scan/nsq063
- Coe, C. L., Kramer, M., Kirschbaum, C., Netter, P., & Fuchs, E. (2002). Prenatal stress diminishes cytokine production after an endotoxin challenge and induces glucocorticoid resistance in juvenile rhesus monkeys. *Journal of Clinical Endocrinology and Metabolism*, 87, 675–681.
- Coe, C. L., Lubach, G. R., & Schneider, M. L. (1999). Neuromotor and socioemotional behavior in the young monkey is presaged by prenatal conditions. In M. Lewis & D. Ramsay (Eds.), *Soothing and stress* (pp. 19–38). Mahwah, NJ: Lawrence Erlbaum Associates.
- Craske, M. G. (2014). Maximizing exposure therapy: An inhibitory learning approach. *Behaviour Research and Therapy*, 58, 10–23.
- Craske, M. G., Treanor, M., Conway, C. C., Zbozinek, T., & Vervliet, B. (2014). Maximizing exposure therapy: An inhibitory learning approach. *Behaviour Research and Therapy*, 58, 10–23.
- Davis, J., Eyre, H., Jacka, F. N., Dodd, S., Dean, O., McEwen, S., ... Berk, M. (2016). A review of vulnerability and risks for schizophrenia: Beyond the two hit hypothesis. *Neuroscience and Biobehavioral Reviews*, 65, 185–194.
- Davis, M. C., Matthews, K. A., & Twamley, E. W. (1999). Is life more difficult on Mars or Venus? A meta-analytic review of sex differences in major and minor life events. *Annals of Behavioral Medicine*, 21, 83–97.
- Del Giudice, M. (2016). Differential susceptibility to the environment: Are developmental models compatible with the evidence from twin studies? *Developmental Psychology*, 52, 1330–1339.
- DeRubeis, R. J., Hollon, S. D., Amsterdam, J. D., Shelton, R. C., Young, P. R., Salomon, R. M., ... Gallop, R. (2005). Cognitive therapy vs medications in the treatment of moderate to severe depression. *Archives of General Psychiatry*, 62, 409–416.
- Dickerson, S. S., & Kemeny, M. E. (2004). Acute stressors and cortisol responses: A theoretical integration and synthesis of laboratory research. *Psychological Bulletin*, 130, 355–391.
- Dimidjian, S., Barrera, M., Martell, C., Muñoz, R. F., & Lewinsohn, P. M. (2011). The origins and current status of behavioral activation treatments for depression. *Annual Review of Clinical Psychology*, 7, 1–38.
- Dudgeon, P., Walker, R., Scrine, C., Shepherd, C., Calma, T., & Ring, I. (2014). *Effective strategies to strengthen the mental health and wellbeing of Aboriginal and Torres Strait Islander people*. Issues paper no. 12, Closing the Gap Clearinghouse. Retrieved from [www.aihw.gov.au/uploadedFiles/ClosingTheGap/Content/Our\\_publications/2014/ctgc\\_ip12.pdf](http://www.aihw.gov.au/uploadedFiles/ClosingTheGap/Content/Our_publications/2014/ctgc_ip12.pdf)
- Duncan, L. E., & Keller, M. C. (2011). A critical review of the first 10 years of candidate gene-by-environment interaction research in psychiatry. *American Journal of Psychiatry*, 168(10), 1041–1049.
- Ellenberger, H. F. (1972). The story of “Anna O”: A critical review with new data. *Journal of the History of the Behavioral Sciences*, 8, 267–279.
- Ellis, A. (1991). The revised ABC’s of rational-emotive therapy (RET). *Journal of Rational-Emotive and Cognitive Behavior Therapy*, 9, 139–172.
- Ellis, A. (1993). Changing rational-emotive therapy (RET) to rational emotive behavior therapy (REBT). *The Behavior Therapist*, 16, 257–258.
- Ellis, A. (1995). Changing rational-emotive therapy (RET) to rational emotive behavior therapy (REBT). *Journal of Rational-Emotive and Cognitive Behavior Therapy*, 13, 85–89.
- Eugenides, J. (2002/2011). *Middlesex* (p. 479). London: Bloomsbury.
- Farina, A. (1976). *Abnormal psychology*. Englewood Cliffs, NJ: Prentice-Hall.
- Fisher, P., & Wells, A. (2009). *Metacognitive therapy: Distinctive features*. East Sussex, UK: Routledge.
- Fraley, R. C., & Shaver, P. R. (2000). Adult romantic attachment: Theoretical developments, emerging controversies, and unanswered questions. *Review of General Psychology*, 4, 132–154.
- Freud, A. (1946/1966). *The ego and mechanisms of defense*. New York: International Universities Press.
- Furtado, M., & Katzman, M. A. (2015a). Examining the role of neuroinflammation in major depression. *Psychiatry Research*, 229, 27–36. doi: 10.1016/j.psychres.2015.06.009
- Furtado, M., & Katzman, M. A. (2015b). Neuroinflammatory pathways in anxiety, posttraumatic stress, and obsessive compulsive disorders. *Psychiatry Research*, 229, 37–48. doi: 10.1016/j.psychres.2015.05.036
- Galletly, C., Castle, D., Dark, F., Humberstone, V., Jablensky, A., Killackey, E., Kulkarni, J., McGorry, P., Nielssen, O., & Tran, N. (2016). *Australian & New Zealand Journal of Psychiatry*, 50, 410–472. doi: 10.1177/0004867416641195
- Galsworthy-Francis, L., & Allan, S. (2014). Cognitive behavioural therapy for anorexia nervosa: A systematic review. *Clinical Psychology Review*, 34, 54–72.
- Gatt, J. M., Burton, K. L. O., Williams, L. M., & Schofield, P. R. (2015). Specific and common genes implicated across major mental disorders: A review of meta-analysis studies. *Journal of Psychiatric Research*, 60, 1–13.
- Gould, S., & Friedman, F. (2016, February 4). Something startling is going on with antidepressant use around the world. *Business Insider*. Retrieved from [www.businessinsider.com/countries-largest-antidepressant-drug-users-2016-2/?r=AU&IR=T](http://www.businessinsider.com/countries-largest-antidepressant-drug-users-2016-2/?r=AU&IR=T)

- Gravel, S., Henn, B. M., Gutenkunst, R. N., Indap, A. R., Marth, G. T., Clark, A. G., et al. (2011). Demographic history and rare allele sharing among human populations. *Proceedings of the National Academy of Sciences*, 108, 11983–11988.
- Gregory, B., Peters, L., & Rapee, R. M. (2016). The self in social anxiety. In M. Kyrios, R. Moulding, G. Doron, S. S. Bhar, M. Nedeljkovic, & M. Mikulincer (Eds.), *The self in understanding and treating psychological disorders* (pp. 91–101). Cambridge University Press.
- Gross, C., Zhuang, X., Stark, K., Ramboz, S., Oosting, R., Kirby, L., Santarelli, L., Beck, S., & Hen, R. (2002). Serotonin 1A receptor acts during development to establish normal anxiety-like behaviour in the adult. *Nature*, 416, 396–400.
- Guidano, V. F. (1991). *The self in process: Towards a post-rationalist cognitive therapy*. New York, NY: Guilford Press.
- Guidano, V. F., & Liotti, G. (1983). *Cognitive processes and emotional disorders: A structural approach to psychotherapy*. New York, NY: The Guilford Press.
- Gutman, D. A., & Nemeroff, C. B. (2003). Persistent central nervous system effects of an adverse early environment: Clinical and preclinical studies. *Physiology and Behavior*, 79, 471–478.
- Halford, W. K., & Bodenmann, G. (2013). Effects of relationship education on maintenance of couple relationship satisfaction. *Clinical Psychology Review*, 33, 512–525. doi:10.1016/j.cpr.2013.02.001
- Halford, W. K., Pepping, C. A., & Petch, J. (2015). The gap between couple therapy research: Efficacy and practice effectiveness. *Journal of Marital and Family Therapy*, 42, 32–44. doi:10.1111/jmft.12120
- Hallahan, L. (2012). Time to stop the forced sterilisation of girls and women with disability. *The Conversation*. Retrieved from <https://theconversation.com/time-to-stop-the-forced-sterilisation-of-girls-and-women-with-disability-9812>
- Harrington, A. (2008). *The cure within: A history of mind-body medicine*. New York: W. W. Norton.
- Harris, R. B., Cormack, D. M., & Stanley, J. (2013). The relationship between socially-assigned ethnicity, health and experience of racial discrimination for Māori: analysis of the 2006/07 New Zealand Health Survey. *BMC Public Health*, 13, 844. doi: 10.1186/1471-2458-13-844
- Hayes, S. C. (2004). Acceptance and commitment therapy, relational frame theory, and the third wave of behavioral and cognitive therapies. *Behavior Therapy*, 35, 639–665.
- Hayes, S. C., Follette, V. M., & Linehan, M. M. (2004). *Mindfulness and acceptance: Expanding the cognitive behavioral tradition*. New York, NY: The Guilford Press.
- Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (2016). *Acceptance and commitment therapy: The process and practice of mindful change (2nd revised edition)*. New York, NY: Guilford Publications.
- Hermans, D., Craske, M. G., Mineka, S., & Lovibond, P. F. (2006). Extinction in human fear conditioning. *Biological Psychiatry*, 60, 361–368.
- Hofmann, S. G., Sawyer, A. T., & Fang, A. (2010). The empirical status of the ‘new wave’ of CBT. *Psychiatric Clinics of North America*, 33, 701–710.
- Hofmann, S. G., Sawyer, A. T., Witt, A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: a meta-analytic review. *Journal of Consulting & Clinical Psychology*, 78, 69–83.
- Hulley, S., Grady, D., Bush, T., Furberg, C., Herrington, D., Riggs, B., & Vittinghoff, E. (1998). Randomized trial of estrogen plus progestin for secondary prevention of coronary heart disease in postmenopausal women. Heart and Estrogen/progestin Replacement Study (HERS) Research Group. *Journal of the American Medical Association*, 280, 605–613.
- Human Genome Project. (2008). *How many genes are in the human genome?* Retrieved from [www.ornl.gov/sci/techresources/Human\\_Genome/faq/genenumber.shtml](http://www.ornl.gov/sci/techresources/Human_Genome/faq/genenumber.shtml)
- Hustvedt, A. (2011). *Medical muses: Hysteria in nineteenth-century Paris*. New York: W. W. Norton.
- Jaffee, S. R., & Price, T. S. (2007). Gene–environment correlations: A review of the evidence and implications for prevention of mental illness. *Molecular Psychiatry*, 12, 432–442. doi:10.1038/sj.mp.4001950
- James, R. (2006). Culture-bound syndromes: Taijin Kyofusho. In Y. Jackson (Ed.), *Encyclopedia of multicultural psychology* (pp. 146–147). Thousand Oaks, CA: Sage Publications. doi:10.4135/9781412952668.n76
- Jazayeri, H., Urry, H. L., & Gross, J. J. (2013). Affective disturbance and psychopathology: An emotion regulation perspective. *Journal of Experimental Psychopathology*, 4, 584–599. doi: 10.5127/jep.030312
- Jorm, A. F., Bourchier, S. J., Cvetkovski, S., & Stewart, G. (2012). Mental health of Indigenous Australians: A review of findings from community surveys. *MJA*, 196, 118–121. doi: 10.5694/mja11.10041
- Karg, K., Burmeister, M., Shedden, K., & Sen, S. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, 68, 444–454.
- Kazdin, A. E., & Weisz, J. R. (1998). Identifying and developing empirically supported child and adolescent treatments. *Journal of Consulting and Clinical Psychology*, 66, 19–36.
- Kendall, P. C., Haaga, D. A. F., Ellis, A., Bernard, M., DiGiuseppe, R., & Kassirnov, H. (1995). Rationale and therapy in the 1990s and beyond: Current status, recent revisions, and research questions. *Clinical Psychology Review*, 15, 169–185.
- Kendler, K. S. (2013). What psychiatric genetics has taught us about the nature of psychiatric illness and what is left to learn. *Molecular Psychiatry*, 18, 1058–1066. doi:10.1038/mp.2013.50
- Kiecolt-Glaser, J. K., & Glaser, R. (2002). Depression and immune function: Central pathways to morbidity and mortality. *Journal of Psychosomatic Research*, 53, 873–876.

- Kirsch, I., Deacon, B. J., Huedo-Medina, T. B., Scoboria, A., Moore, T. J., & Johnson, B. T. (2008). Initial severity and antidepressant benefits: A meta-analysis of data submitted to the food and drug administration. *PLOS Medicine*, 5(2), e45. doi: 10.1371/journal.pmed.0050045.
- Kring, A. M. (2000). Gender and anger. In A. H. Fischer (Ed.), *Gender and Emotion* (pp. 211–231). Cambridge, UK: Cambridge University Press.
- Kuhn, T. S. (1962/1970). *The structure of scientific revolutions*. Chicago: University of Chicago Press.
- Kunst-Wilson, W. R., & Zajonc, R. B. (1980). Affective discrimination of stimuli that cannot be recognized. *Science*, 207, 557–558.
- Kyrios, M. (2014). Anxiety disorders in primary care. In P. Emmelkamp & T. Ehring (Eds.), *International handbook of anxiety disorders: Theory, research and practice* (pp. 58–68). Wiley-Blackwell.
- Kyrios, M. (2016). The self in psychological disorders: An introduction. In M. Kyrios, R. Moulding, G. Doron, S. S. Bhar, M. Nedeljkovic, & M. Mikulincer (Eds.), *The self in understanding and treating psychological disorders* (pp. 1–7). Cambridge University Press.
- Kyrios, M., Moore, S., Hackworth, N., Buzwell, S., Crafti, N., Critchley, C., & Hardie, E. (2009). The influence of depression and anxiety in outcomes following intervention for pre-diabetes. *Medical Journal of Australia*, 190, S81–S85.
- Kyrios, M., Moulding, R., Doron, G., Bhar, S. S., Nedeljkovic, M., & Mikulincer, M. (Eds.) (2016). *The self in understanding and treating psychological disorders*. Cambridge: Cambridge University Press.
- Kyrios, M., & Thomas, N. (2014). Psychology and the Internet: Where are we and where to from here? *InPsych*, 36(3), 7–10.
- Lavoie, J. G., Kornelsen, D., Wylie, L., Mignone, J., Dwyer, J., Boyer, Y., . . . O'Donnell, K. (2016). Responding to health inequities: Indigenous health system innovations. *Global Health, Epidemiology and Genomics*, 1, e14, 1–9. doi:10.1017/ghg.2016.12
- Lavoie, K. L., Miller, S. B., Conway, M., & Fleet, R. P. (2001). Anger, negative emotions, and cardiovascular reactivity during interpersonal conflict in women. *Journal of Psychosomatic Research*, 15, 503–512.
- Leahy, R. L. (2003). *Cognitive therapy techniques: A practitioner's guide*. New York: Guilford Press.
- Lau, J. Y. F., & Eley, T. C. (2008). Attributional style as a risk marker of genetic effects for adolescent depressive symptoms. *Journal of Abnormal Psychology*, 117, 849–859. doi: 10.1037/a0013943
- Lau, J. Y. F., Hilbert, K., Goodman, R., Gregory, A. M., Pine, D. S., Viding, E. M., & Eley, T. C. (2012). Investigating the genetic and environmental bases of biases in threat recognition and avoidance in children with anxiety problems. *Biology of Mood and Anxiety Disorders*, 2, 12. doi: 10.1186/2045-5380-2-12
- Leske, S., Harris, M. G., Charlson, F. J., Ferrari, A. J., Baxter, A. J., Logan, J. M., . . . Whiteford, H. (2016). Systematic review of interventions for Indigenous adults with mental and substance use disorders in Australia, Canada, New Zealand and the United States. *Australian & New Zealand Journal of Psychiatry*, 50, 1040–1054. doi: 10.1177/0004867416662150
- Lichtenstein, P., Yip, B. H., Bjork, C., et al. (2009). Common genetic determinants of schizophrenia and bipolar disorder in Swedish families: A population-based study. *Lancet*, 373, 234–239.
- Linehan, M. M. (1993). *Cognitive-behavioral treatment of borderline personality disorder*. New York, NY: Guilford Press.
- Luyten, P., Mayes, L., Fonagy, P., Target, M., & Blatt, S. (Eds.) (2015). *Handbook of contemporary psychodynamic approaches to psychopathology*. New York: Guilford, pp. 199–215.
- MacLeod, C., & Clarke, P. J. F. (2015). The attentional bias modification approach to Anxiety intervention. *Clinical Psychological Science*, 3, 58–78. doi: 10.1177/2167702614560749
- Maier, S. F., & Watkins, L. R. (1998). Cytokines for psychologists: Implications of bidirectional immune-to-brain communication for understanding behavior, mood, and cognition. *Psychological Review*, 105, 83–107.
- Main, M., Kaplan, K., & Cassidy, J. (1985). Security in infancy, childhood, and adulthood: A move to the level of representation. *Monographs of the Society for Research in Child Development*, 50 (1–2, Serial No. 209).
- Manson, J. E., Chlebowski, R. T., Stefanick, M. L., Aragaki, A. K., Rossouw, J. E., Prentice, R. L., et al. (2013). Menopausal hormone therapy and health outcomes during the intervention and extended poststopping phases of the Women's Health Initiative randomized trials. *Journal of the American Medical Association*, 310(13), 1353–1368.
- Mataix-Cols, D., Billotti, D., Fernández de la Cruz, L., & Nordsletten, A. E. (2013). The London field trial for hoarding disorder. *Psychological Medicine*, 43, 837–847.
- Matthews, K. A., Owens, J. F., Kuller, L. H., Sutton-Tyrrell, K., & Jansen-McWilliams, L. (1998). Are hostility and anxiety associated with carotid atherosclerosis in health postmenopausal women? *Psychosomatic Medicine*, 60, 633–638.
- McGorry, P. D., Yung, A. R., Phillips, L. J., Yuen, H. P., Francey, S., Cosgrave, E. M., et al. (2002). Randomized controlled trial of interventions designed to reduce the risk of progression to first-episode psychosis in a clinical sample with subthreshold symptoms. *Archives of General Psychiatry*, 59, 921–928.
- Melamed, B. G., & Siegel, L. J. (1975). Reduction of anxiety in children facing hospitalization and surgery by use of filmed modeling. *Journal of Consulting and Clinical Psychology*, 43, 511–521.
- Mendelsohn, F. (2013). Understanding the brain and mind: science's final frontier? *The Conversation*. Retrieved from <https://theconversation.com/understanding-the-brain-and-mind-sciences-final-frontier-18331>
- Miklowitz, D. J., & Taylor, D. O. (2005). Family-focused treatment of the suicidal bipolar patient. Unpublished manuscript.
- Miklowitz, D. J., George, E. L., Richards, J. A., Simoneau, T. L., & Suddath, R. L. (2003). A randomized study of family-focused psychoeducation and pharmacotherapy in the outpatient management of bipolar disorder. *Archives of General Psychiatry*, 60, 904–912.

- Ministry of Health. (2008). *A portrait of health: Key results of the 2006/07 New Zealand Health Survey*. Wellington: Ministry of Health.
- Moniz, E. (1936). *Tentatives operatoires dans le traitement de certaines psychoses*. Paris: Mason.
- Moraes, F., & Goes, A. (2016). A decade of human genome project conclusion: Scientific diffusion about our genome knowledge. *Biochemistry and Molecular Biology Education*, 44, 215–223.
- Murphy, J. (1976). Psychiatric labeling in cross-cultural perspective. *Science*, 191, 1019–1028.
- Murray, G. (Ed.) (2012). *A critical introduction to DSM*. New York, NY: Nova Science Publishers.
- Myers, K. M., & Davis, M. (2007). Mechanisms of fear extinction. *Molecular Psychiatry*, 12, 120–150. doi:10.1038/sj.mp.4001939.
- Nedeljkovic, M., Moulding, R., Foroughi, E., Kyrios, M., & Doron, G. (2011). Cultural issues in understanding and treating obsessive-compulsive and spectrum disorders. In G. Steketee (Ed.), *Oxford Handbook of Obsessive Compulsive and Spectrum Disorders* (pp. 496–520). New York, NY: Oxford University Press.
- Neisser, U. (1976). *Cognition and reality*. San Francisco: Freeman.
- Nestler, E. J., Peña, C. J., Kundakovic, M., Mitchell, A., & Akbarian, S. (2016). Epigenetic basis of mental illness. *The Neuroscientist*, 22, 447–463. doi: 10.1177/1073858415608147
- O'Donohue, W. (1993). The spell of Kuhn on psychology: An exegetical elixir. *Philosophical Psychology*, 6, 267–287.
- O'Keefe, J. (2014). Nobel Prize Banquet Speech, 10 December 2014. Retrieved from [https://www.nobelprize.org/nobel\\_prizes](https://www.nobelprize.org/nobel_prizes)
- Öst, L. G. (2008). Efficacy of the third wave of behavioral therapies: A systematic review and meta-analysis. *Behaviour Research and Therapy*, 46, 296–321.
- Penn, D. L., Chamberlin, C., & Mueser, K. T. (2003). The effects of a documentary film about schizophrenia on psychiatric stigma. *Schizophrenia Bulletin*, 29, 383–391.
- Pescosolido, B. A., Martin, J. K., Long, J. S., Medina, T. R., Phelan, J. C., & Link, B. G. (2010). 'A disease like any other?' A decade of change in public relations to schizophrenia, depression and alcohol dependence. *American Journal of Psychiatry*, 167, 1321–1330.
- Pietromonaco, P. R., & Barrett, L. F. (1997). Working models of attachment and daily social interactions. *Journal of Personality and Social Psychology*, 73, 1409–1423.
- Plomin, R. (1999). Genetics and general cognitive ability. *Nature*, 402, C25–C29.
- Plomin, R., DeFries, J. C., Craig, I. W., & McGuffin, P. (2003). *Behavioral genetics in the postgenomic era*. Washington, DC: APA Books.
- Plomin, R., DeFries, J. C., Knopik, V. S., & Neiderhiser, J. M. (2013). *Behavioral genetics* (6th ed.). New York, NY: Worth.
- Polderman, T. J., Benyamin, B., de Leeuw, C. A., Sullivan, P. F., van Bochoven, A., Visscher, P. M., & Posthuma, D. (2015). Meta-analysis of the heritability of human traits based on fifty years of twin studies. *Nature Genetics*, 47, 702–709. <http://dx.doi.org/10.1038/ng.3285>
- Poroch, N., Arabena, K., Tongs, J., Larkin, S., Fisher, J., & Henderson, G. (2009). *Spirituality and Aboriginal people's social and emotional wellbeing: A review*. Cooperative Research Centre for Aboriginal Health Discussion Paper Series: No. 11.
- Purdie, N., Dudgeon, P., & Walker, R. (Eds.) (2010). *Working together: Aboriginal and Torres Strait Islander mental health and wellbeing principles and practice*. Canberra: Commonwealth of Australia.
- Reynolds, J., Griffiths, K. M., Cunningham, J. A., Bennett, K., & Bennett, A. (2015). Clinical practice models for the use of e-mental health resources in primary health care by health professionals and peer workers: A conceptual framework. *JMIR Mental Health*, 2(1):e6. doi:10.2196/mental.4200
- Ridley, M. (2003). *Nature via nurture: Genes, experience, and what makes us human*. Great Britain: HarperCollins.
- Rogers, C. (1953). *Client-centered therapy: Its current practice, implications and theory*. London: Constable.
- Roughton, E. C., Schneider, M. L., Bromley, L. J., & Coe, C. L. (1998). Maternal activation during pregnancy alters neurobehavioral state in primate infants. *American Journal of Occupational Therapy*, 52, 90–98.
- Rutledge, T., Reis, S. E., Olson, M., Owens, J., Kelsey, S. F., Pepine, C. J., et al. (2001). Psychosocial variables are associated with atherosclerosis risk factors among women with chest pain: The WISE study. *Psychosomatic Medicine*, 63, 282–288.
- Rutter, M., & Silberg, J. (2002). Gene-environment interplay in relation to emotional and behavioral disturbance. *Annual Review of Psychology*, 53, 463–490.
- Sedikides, C., & Brewer, M. B. (Eds.). (2001). *Individual self, relational self, collective self*. Philadelphia, PA: Psychology Press.
- Segal, Z. V., Williams, J. M. G., & Teasdale, J. D. (2013). *Mindfulness-based cognitive therapy for depression, second edition*. New York: Guilford Press.
- Segerstrom, S. C., & Miller, G. E. (2004). Psychological stress and the immune system: A meta-analytic study of 30 years of inquiry. *Psychological Bulletin*, 130, 601–630.
- Selling, L. S. (1940). *Men against madness*. New York: Greenberg.
- Shenk, D. (2010). *The genius is in all of us: Why everything you've been told about genetics, talent, and IQ is wrong*. New York: Doubleday.
- Singer, J. A. (2004). Narrative identity and meaning making across the adult lifespan: An introduction. *Journal of Personality*, 72, 437–459.
- Slade, T., Johnston, A., Oakley Browne, M. A., Andrews, G., & Whiteford, H. (2009). 2007 National Survey of Mental Health and Wellbeing: methods and key findings. *Australian And New Zealand Journal Of Psychiatry*, 43, 594–605.

- State, M. W., & Geschwind, D. H. (2015). Leveraging genetics and genomics to define the causes of mental illness. *Biological Psychiatry*, 77, 3–5.
- Sullivan, P. F., Kendler, K. S., & Neale, M. C. (2003). Schizophrenia as a complex trait — Evidence from a meta-analysis of twin studies. *Archives of General Psychiatry*, 60, 1187–1192.
- Suls, J., & Bunde, J. (2005). Anger, anxiety, and depression as risk factors for cardiovascular disease: The problems and implications of overlapping affective dispositions. *Psychological Bulletin*, 131, 260–300.
- Tarumi, S., Ichimiya, A., Yamada, S., Umesue, M., & Kuroki, T. (2004). Taijin Kyofusho in university students: Patterns of fear and predispositions to the offensive variant. *Transcultural Psychiatry*, 41, 533–46. doi:10.1177/1363461504047933.
- Thase, M. E., Kingdon, D., & Turkington, D. (2014). The promise of cognitive behavior therapy for treatment of severe mental disorders: A review of recent developments. *World Psychiatry*, 13, 244–250.
- Thoits, P. A. (1985). Self-labeling processes in mental illness: The role of emotional deviance. *American Journal of Sociology*, 92, 221–249.
- Thornicroft, G., Rose, D., Kassam, A., & Sartorius, N. (2007). Stigma: ignorance, prejudice or discrimination? *British Journal of Psychiatry*, 190, 192–193.
- Turkheimer, E. (1998). Heritability and biological explanation. *Psychological Review*, 105, 782–791.
- Turkheimer, E., Haley, A., Waldron, M., D'Onofrio, B., & Gottesman, I. I. (2003). Socioeconomic status modifies the heritability of IQ in young children. *Psychological Science*, 6, 623–628.
- Turkheimer, E. (2000). Three laws of behavior genetics and what they mean. *Current Directions in Psychological Science*, 9, 160–164.
- Turkheimer, E., Pettersson, E., & Horn, E. E. (2014). A phenotypic null hypothesis for the genetics of personality. *Annual Review of Psychology*, 65, 515–540.
- Turner, J. C. (1985). Social categorization and the self-concept: A social cognitive theory of group behaviour. In E. J. Lawler (Ed.), *Advances in group processes* (vol. 2, pp. 77–122). Greenwich, CT: JAI Press.
- Turner, J. C., Hogg, M. A., Oakes, P. J., Reicher, S. D., & Wetherell, M. S. (1987). *Rediscovering the social group: A self-categorization theory*. Oxford: Blackwell.
- Turner, J. C., Oakes, P. J., Haslam, S. A., & McGarty, C. (1994). Self and collective: Cognition and social context. *Personality and Social Psychology Bulletin*, 20, 454–463. doi:10.1177/0146167294205002
- United Nations. (2015). *Serious concerns about human rights violations against Australians with disability*. Retrieved from <http://wwda.org.au/united-nations-serious-concerns-about-human-rights-violations-against-australians-with-disability>
- Van der Gaag, M., Smit, F., Bechdolf, A., French, P., Linszen, D.H., Yung, A.R., McGorry, P., & Cuijpers, P. (2013). Preventing a first episode of psychosis: Meta-analysis of randomized controlled prevention trials of 12 month and longer-term follow-ups. *Schizophrenia Research*, 149, 56–62.
- Vassallo, S., & Sanson, A. (Eds) (2013). *The Australian Temperament Project: The first 30 years*. Melbourne, Australia: Australian Institute of Family Studies.
- Wakefield, J. C. (1992). Disorder as dysfunction: A conceptual critique of DSM-III-R's definition of mental disorder. *Psychological Review*, 99, 232–247.
- Wakefield, J. C. (1999). Philosophy of science and the progressiveness of DSM's theory—neutral nosology: Response to Follette and Houts, Part 1. *Behaviour Research and Therapy*, 37, 963–969.
- Waszczuk, M. A., Zavos, H. M. S., Antonova, E., Haworth, C. M., Plomin, R., & Eley, T. (2015). A multivariate twin study of trait mindfulness, depressive symptoms, and anxiety sensitivity. *Depression and Anxiety*, 32, 254–261.
- Waters, T. L., & Barrett, P. M. (2000). The role of the family in childhood obsessive-compulsive disorder. *Clinical Child and Family Psychology Review*, 3, 173–84. doi:10.1023/A:1009551325629
- Watkins, P. C. (2002). Implicit memory bias in depression. *Cognition and Emotion*, 16, 381–402.
- Weisman, C. S., & Teitelbaum, M. A. (1985). Physician gender and the physician–patient relationship: Recent evidence and relevant questions. *Social Science and Medicine*, 20, 1119–1127.
- Wesley Mission (2007). *Living with mental illness: Attitudes, experiences and challenges*. Wesley Mission.
- Westen, D. (1998). The scientific legacy of Sigmund Freud: Toward a psychodynamically informed psychological science. *Psychological Bulletin*, 124, 333–371.
- Whisman, M. A., & Uebelacker, L. A. (2006). Impairment and distress associated with relationship discord in a national sample of married or cohabiting adults. *Journal of Family Psychology*, 20, 369–377.
- Whitaker, R. (2002). *Mad in America*. Cambridge, MA: Perseus.
- White, M. (2007). *Maps of narrative practice*. New York, NY: Norton Professional Books.
- Wichers, M. C., Myin-Germeys, I., Jacobs, N., Peeters, F., Kenis, G., Derom, C., . . . van Os, J. (2007). Evidence that moment-to-moment variation in positive emotions buffer genetic risk for depression: a momentary assessment twin study. *Acta Psychiatrica Scandinavica*, 115, 451–457.
- Wolpe, J. (1954). Reciprocal inhibition as the main basis of psychotherapeutic effects. *Archives of Neurology and Psychiatry*, 72, 205–226. doi:10.1001/archneurpsyc.1954.02330020073007
- Writing Group for the Women's Health Initiative Investigators. (2002). Risks and benefits of estrogen plus progestin in healthy postmenopausal women. Principal results from the Women's Health Initiative randomized controlled trial. *Journal of the American Medical Association*, 288, 321–333.

Yücel, M., Harrison, B. J., Wood, S. J., Fornito, A., Wellard, R. M., Pujol, J., . . . Pantelis, C. (2007). Functional and biochemical alterations of the medial frontal cortex in obsessive-compulsive disorder. *Archives of General Psychiatry*, 64, 946–955.

Zhang, T. Y., & Meaney, M. J. (2010). Epigenetics and the environmental regulation of the genome and its function. *Annual Review of Psychology*, 61, 439–466.

Zubin, J., & Spring, B. (1977). Vulnerability: A new view of schizophrenia. *Journal of Abnormal Psychology*, 86, 103–126.

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## CHAPTER 2

# Diagnosis and assessment

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 2.1** distinguish the different types of reliability and validity
  - 2.2** identify the basic features, historical changes, strengths and weaknesses of the DSM
  - 2.3** describe the goals, strengths and weaknesses of psychological approaches to assessment
  - 2.4** describe the goals, strengths and weaknesses of neurobiological approaches to assessment
  - 2.5** discuss the ways in which culture and ethnicity impact diagnosis and assessment.
-

## OPENING SCENARIO

After six months of treatment Jack's medical practitioner could not find the cause of his ongoing pain. Jack, a 21-year-old university student, first sought medical attention for pain in his ankle after falling while running. Jack's doctor found no obvious signs of injury. Over time, he complained of increasing pain in his ankle as well as pain in his knees, wrists, elbows and shoulders. He said his injuries were the result of falls while running or riding his bike. His doctor could find no obvious signs of injury and was concerned that Jack's pain was not responding to treatment.

When Jack found his pain unbearable, his mother called an ambulance. At the hospital, the doctors could not find a physical cause of his pain. Jack told the doctors that he believed his pain was the result of electromagnetic impulses generated by a device that had been implanted in his body. According to Jack, this device had been implanted by the government shortly after his birth. Jack believed that many babies had these devices implanted so the government could monitor the movements of its citizens. He claimed the electromagnetic impulses caused his falls and his pain.

Jack had come to believe that his mother had consented to the device being implanted and had known about it all along. He was unsure who he could trust and asked if he could have the implant removed. Jack became distressed when his mother told him he was crazy to think this and when the doctors told him there was no sign of an implant. He concluded they all must be working for the government.

### QUESTIONS

1. What needs to happen next before Jack can be treated?
2. What possible diagnoses could be considered?
3. Imagine you are Jack. What emotions would you be experiencing while at the hospital?

## Introduction

Diagnosis and assessment are the critically important 'first steps' in the study and treatment of psychopathology. In the case of Jack, a clinician may begin treatment by determining whether he meets the diagnostic criteria for a mood disorder, schizophrenia or perhaps a substance use disorder. Diagnosis can be the first major step in good clinical care. Having a correct **diagnosis** will allow the clinician to describe the baseline of Jack's symptoms and functioning. Diagnosis provides information about the possible causes of Jack's symptoms and the direction of treatment. Assessment and accurate diagnosis are important aspects of good clinical care. More broadly, imagine that your doctor told you, 'There is no diagnosis for what you have'. This scenario could lead to confusion and feelings of helplessness because without a diagnosis treatment cannot be accurately selected. Rather than this alarming scenario, hearing a diagnosis can provide relief in several different ways. Often, a diagnosis can help a person begin to understand why certain symptoms are occurring, which can be a huge relief. Many disorders, such as depression and anxiety, are extremely common; knowing that his or her diagnosis is common can also help a person feel less unusual.

Diagnosis enables clinicians and scientists to communicate accurately with one another about cases or research. Without agreed-on definitions and categories, different scientists and clinicians would be unable to understand each other.

Diagnosis is important for research on causes and treatments of symptoms. Sometimes researchers discover unique causes and treatments associated with a certain set of symptoms. For example, autism spectrum disorder (ASD) was only recognised in the *Diagnostic and Statistical Manual of Mental Disorders* in 1980. Since that time, research on the causes and treatments of ASD has grown exponentially.

To help make the correct diagnosis, clinicians and researchers use a variety of assessment procedures, beginning with a clinical interview. Broadly speaking, all clinical assessment procedures are more or less formal ways of finding out what is wrong with a person, what may have caused problems, and what can be done to improve the person's condition. Assessment procedures can help in making a diagnosis and they can also provide information beyond a diagnosis. Indeed, a diagnosis is only a starting point. In the case of Jack, for example, many other questions remain to be answered. Why does Jack believe

his pain is caused by a device implanted in his body by the government? What can be done to help him trust the people who are there to help him? Has he performed up to his intellectual potential in school and in his studies at university? What obstacles might interfere with treatment? These are also the types of questions that mental health professionals address in their assessments.

In this chapter, we will describe the official diagnostic system used by many mental health professionals, as well as the strengths and weaknesses of this system. We will then turn to a discussion of the most widely used assessment techniques, including interviews, psychological assessment and neurobiological assessment. We then conclude the chapter with an examination of a sometimes neglected aspect of assessment, the role of cultural bias. Before considering diagnosis and assessment in detail, however, we begin with a discussion of two concepts that play a key role in diagnosis and assessment: reliability and validity.

## 2.1 Cornerstones of diagnosis and assessment

**LEARNING OUTCOME 2.1** Distinguish the different types of reliability and validity.

The concepts of reliability and validity are the cornerstones of any diagnostic or assessment procedure. Without them, the usefulness of our methods is seriously limited. That said, these two concepts are quite complex. Here, we provide a general overview.

### Reliability

**Reliability** refers to consistency of measurement. An example of a reliable measure would be a wooden ruler, which produces the same value every time it is used to measure an object. In contrast, an unreliable measure would be a flexible, elastic-like ruler whose length changes every time it is used. Several types of reliability exist, and here we will discuss the types that are most central to assessment and diagnosis.

**Interrater reliability** refers to the degree to which two independent observers agree on what they have observed. To take an example from cricket, two umpires may or may not agree that a ball is out. In psychology, interrater reliability refers to the extent two clinicians observing the behaviour of the same patients agree on a diagnosis.

**Test-retest reliability** measures the extent to which people being observed twice or taking the same test twice, perhaps several weeks or months apart, receive similar scores. This kind of reliability makes sense only when we can assume that the people will not change appreciably between test sessions on the underlying variable being measured; a prime example of a situation in which this type of reliability is typically high is in evaluating intelligence tests. When a person takes an intelligence test it is expected that their performance will not significantly differ if they take the test twice. Of course, the timing of the re-test is important; if the duration between tests is too brief, the person may recall information from the first test administration. If the duration is too long the person may have changed in ways that could impact on test performance. Further, we cannot expect people to be in the same mood at a baseline and a follow-up assessment four weeks later and thus the test-retest reliability of a mood measure would likely be low.

Reliability is an essential property of all assessment procedures. One means of establishing reliability is to determine whether different judges agree, as happens when two umpires witness the same event in a cricket match.



Sometimes psychologists use two forms of a test rather than give the same test twice, perhaps when there is concern that test takers will remember their answers from the first round of taking the test and aim merely to be consistent. This approach enables the tester to determine **alternate-form reliability**, the extent to which scores on the two forms of the test are consistent.

Finally, **internal consistency reliability** assesses whether the items on a test are related to one another. For example, one would expect the items on an anxiety questionnaire to be interrelated or to correlate with one another, if they truly measure anxiety. A person who reports a dry mouth in a threatening situation would be expected to report increases in muscle tension as well, since both are common characteristics of anxiety.

Reliability is typically measured on a scale from 0 to 1.0; the closer the number is to 1.0, the higher the reliability. For all types of reliability, the higher the number, the better the reliability. For example, a test with an internal consistency reliability of .65 is only moderately reliable; a different test with an internal consistency reliability of .91 is very reliable.

## Validity

**Validity** is a complex concept, generally related to whether a measure measures what it is supposed to measure. For example, if a questionnaire is supposed to measure a person's hostility, does it do so? Before we describe types of validity, it is important to note that validity is related to reliability — unreliable measures will not have good validity. Because an unreliable measure does not yield consistent results (recall our example of a ruler whose length is constantly changing), it will not relate very strongly to other measures. For example, an unreliable measure of coping is not likely to relate well to how a person adjusts to stressful life experiences. Reliability, however, does not guarantee validity. Height can be measured very reliably, but height would not be a valid measure of anxiety.

**Content validity** refers to whether a measure adequately samples the domain of interest. For example, a test to assess social anxiety ought to include items that cover feelings of anxiety in different social situations. It has excellent content validity because it contains questions about all the symptoms associated with social anxiety (see a later chapter). For certain uses, though, the test might have poor content validity. The test doesn't cover questions about anxiety in non-social situations and if one were trying to assess specific phobias about heights or snakes, this test would have poor content validity.

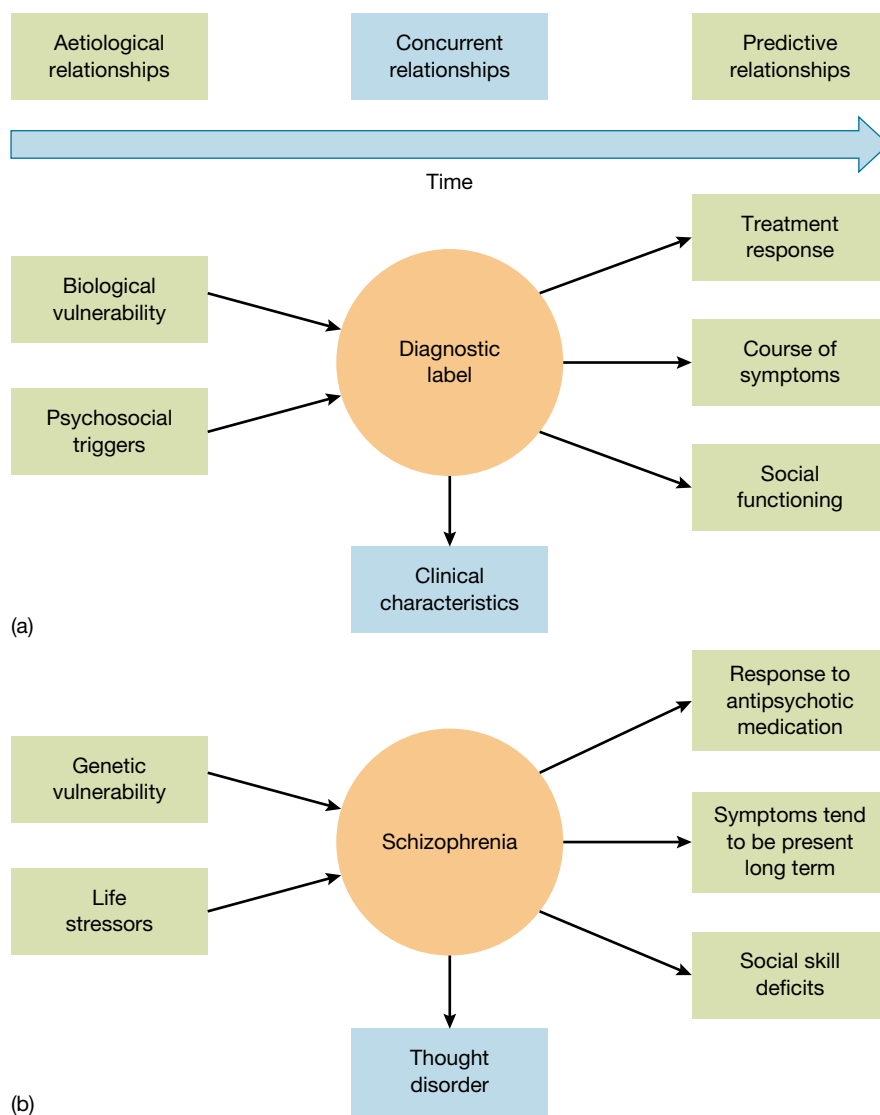
**Criterion validity** is evaluated by determining whether a measure is associated in an expected way with some other measure (the criterion). If both variables are measured at the same point in time, the resulting validity is referred to as **concurrent validity**. For example, later we will describe a measure of the overly negative thoughts that are believed to play an important role in depression. Criterion validity for this measure of negative thoughts could be established by showing that people with depression score higher on the test than do people without depression. In this example, the criterion is the diagnosis of depression. Another way to show concurrent validity is by showing that the measure of negative thoughts is correlated with a measure of depression symptoms.

Alternatively, criterion validity can be assessed by evaluating the ability of the measure to predict some other variable that is measured at some point in the future, often referred to as **predictive validity**. For example, IQ tests were originally developed to predict future school performance. Similarly, a measure of negative thoughts could be used to predict the development of depression in the future. In summary, concurrent and predictive validity are both types of criterion validity.

**Construct validity** is a more complex concept. It is relevant when we want to interpret a test as a measure of some characteristic or construct that is not observed simply or overtly (Cronbach & Meehl, 1955; Hyman, 2002). A construct is an inferred attribute, such as anxiousness or distorted cognition. Consider an anxiety-proneness questionnaire as an example. If the questionnaire has construct validity, people who obtain different scores on our test really will differ in anxiety proneness. Just because the items seem to be about the tendency to become anxious ('I find that I become anxious in many situations'), it is not certain that the test is a valid measure of the construct of anxiety proneness.

As shown in figure 2.1, construct validity is evaluated by looking at a wide variety of data from multiple sources (compare this to criterion validity, where a test is typically evaluated against just one other piece of data). For example, people diagnosed as having an anxiety disorder and people without such a diagnosis could be compared on their scores on our self-report measure of anxiety proneness. The self-report measure would achieve some construct validity if the people with anxiety disorders scored higher than the people without anxiety disorders. Greater construct validity would be achieved by showing that the self-report measure was related to other measures thought to reflect anxiety, such as observations of fidgeting and trembling, and physiological indicators, such as increased heart rate and rapid breathing. When the self-report measure is associated with these multiple measures (diagnosis, observational indicators, physiological measures), its construct validity is increased.

**FIGURE 2.1** Construct validity: an example of the types of information a diagnosis might help predict



More broadly, construct validity is related to theory. For example, we might hypothesise that being prone to anxiety is in part caused by a family history of anxiety. We could then obtain further evidence

for the construct validity of our questionnaire by showing that it relates to a family history of anxiety. At the same time, we would also have gathered support for our theory of anxiety proneness. Thus, construct validation is an important part of the process of theory testing.

### CLINICAL CASE

#### Roxanne

Roxanne is a middle-aged woman who was brought to the emergency department of her local hospital by the police. They had found her running through a crowded street, laughing loudly and running into people. Her clothes were dirty and torn. When they questioned her, she was speaking rapidly and her thoughts were hard to follow. At the emergency department, she wrestled free of the police and began running down the hallway. She knocked over two staff members during her flight, while bellowing at the top of her lungs, 'I am the resurrection! Come follow me!' Police brought her back to the exam room and the staff began to form hypotheses. Clearly, she was full of energy. Had she been through some trauma? She believed she had special religious powers — could this be a delusion? Unfortunately, the staff was unable to gain much information from an interview due to her rapid and incoherent speech. Roxanne sat restlessly, occasionally laughing and shouting; treatment could not proceed without understanding the reason for her unusual behaviour. When efforts to calm Roxanne failed, police helped the staff to contact family members, who were relieved to hear that Roxanne was safe. She had disappeared from home the day before. Family members described a long history of bipolar disorder (formerly known as manic depression) and they reported having been concerned for the past couple of weeks because Roxanne had stopped taking medications for both her bipolar disorder and her high blood pressure. Treatment was able to proceed based on the idea that Roxanne was experiencing a new manic episode of her long-standing bipolar disorder.

#### QUESTIONS

1. Based on the available information, is it easy for the clinicians to conclude that Roxanne is experiencing a new manic episode? Do you think there are other possible diagnoses the clinicians may have missed?
2. What else might a comprehensive assessment of Roxanne's symptoms and experience need to consider?
3. Imagine you are Roxanne. What emotions would you be experiencing while at the hospital?

Clinicians and researchers in psychology have an ethical responsibility to use assessment tools that are valid and reliable measures. If clinicians and researchers use assessment tools that have questionable validity and reliability, inaccurate conclusions will be drawn from the test results. This could result in inaccurate recommendations that could have disastrous consequences for patients. Sometimes researchers and clinicians develop assessment tools and therefore need to be aware of validity and reliability.

### RESEARCH EXAMPLE

#### 'People like numbers'

In a study titled 'People like numbers: a descriptive study of cognitive assessment methods in clinical practice for Aboriginal Australians in the Northern Territory', Dingwall, Pinkerton, and Lindeman (2013) interviewed 22 clinicians working with Indigenous clients in central and northern Australia with the aim of understanding how clinicians approach cognitive assessment with Indigenous clients. This investigation is important because there are currently no well-researched cognitive assessment tools with established validity and reliability for use with this population. Also, clinicians receive little guidance on how to appropriately assess Indigenous clients. Dingwall and colleagues found that clinicians use a range of standardised tests that were modified for use with this client population as well as informal ways of assessing cognitive functioning such as observation. This study revealed that cognitive tests developed specifically for Indigenous clients are urgently needed as well as training on how to approach assessment with this client population. These are crucial for the fair assessment and interpretation of test scores.

#### QUESTION

Can valid and fair conclusions be made about the cognitive functioning of Indigenous people using the approaches described by the clinicians in the research of Dingwall et al. (2013)?

## 2.2 The diagnostic system of the American Psychiatric Association: DSM-5

**LEARNING OUTCOME 2.2** Identify the basic features, historical changes, strengths and weaknesses of the DSM.

In this section, we focus on the diagnostic system used by many mental health professionals, the *Diagnostic and Statistical Manual of Mental Disorders (DSM)*. In 1952, the American Psychiatric Association published its first *Diagnostic and Statistical Manual (DSM)*. The publication of the DSM was informed by earlier systems of classification (see focus on discovery 2.1) and it has been revised five times since 1952. The fifth edition of the DSM, referred to as DSM-5, was released in 2013 ([www.dsm5.org](http://www.dsm5.org)). In this chapter, we will review the major features of DSM-5 and then we will outline some strengths and criticisms of the DSM as well as of diagnosis in general. Because DSM-5 is new, we will note major differences between the DSM-IV-TR and the DSM-5 throughout the chapters of this text.

Each version of the DSM has included improvements. Beginning with the third edition of DSM and continuing today, an effort was made to create more reliable and valid diagnostic categories. Two major innovations were introduced in DSM-III that have been retained by each edition since.

1. Specific diagnostic criteria — the symptoms for a given diagnosis — are spelled out precisely and clinical symptoms are defined in a glossary. Table 2.1 compares the descriptions of a manic episode given in DSM-II to those given in DSM-5. Notice how DSM-5 is much more detailed and concrete.
2. The characteristics of each diagnosis are described much more extensively in DSM-5 than they were in DSM-II. For each disorder there is a description of diagnostic features as well as associated features, such as laboratory findings (e.g., enlarged ventricles in schizophrenia) and results from physical exams (e.g., electrolyte imbalances in people who have eating disorders). Next, a summary of the research literature provides information about age of onset, course, prevalence, risk and prognosis factors, cultural and gender factors and differential diagnosis (i.e., how to distinguish similar diagnoses from each other).

**TABLE 2.1** Mania in DSM-II versus DSM-5

### DSM-II (1968)

Mania was described in DSM-II in just a short paragraph. The paragraph described mania with five symptoms (elevated mood, irritable mood, racing thoughts, rapid talking and rapid movement). There was no mention of how many symptoms were needed to meet the criteria for a manic episode.

### DSM-5 (2013)

DSM-5 provides a list of symptoms with descriptive detail for each symptom. The symptom description is divided into four parts, which we briefly summarise here. In the actual DSM-5, there is more detail for all four parts.

1. The first part describes the mood and energy symptoms for mania. A person must show an elevated or irritable mood as well as a great deal of goal-directed behaviour and energy. These mood and energy symptoms must last for at least one week.
2. In addition to the mood and energy symptoms, a person must also have three or more symptoms, including racing thoughts, rapid talking, very little need for sleep, very high self-esteem or grandiose ideas about the self, difficulty maintaining focused attention, participating in behaviours that can get a person into trouble (e.g., excessive spending or sexual encounters) and excessive movements.
3. All of these symptoms make it very difficult to work or have healthy social relationships. Some people may need to be in the hospital to protect themselves or others.
4. The last part of the diagnosis is a 'rule out' section to clarify whether or not the symptoms cannot be better accounted for by something else, such as another disorder or the effects of a drug (legal or not).

## A history of classification and diagnosis

By the end of the nineteenth century, medical diagnostic procedures were improving as physicians began to understand the advantages of tailoring treatments to different illnesses. During the same period, other sciences, such as botany and chemistry, advanced after classification systems were developed. Impressed by these successes, investigators of psychological disorders also sought to develop classification schemes. Unfortunately, progress in classifying psychological disorders did not come easily.

### Early efforts at classification of psychological disorders

Emil Kraepelin (1856–1926) authored an influential early classification system in his textbook of psychiatry first published in 1883. His classification system attempted to definitively establish the biological nature of psychological disorders. Kraepelin noted that certain symptoms clustered together as a *syndrome*. He labelled a set of syndromes and hypothesised that each had its own biological cause, course and outcome. Even though effective treatments had not been identified, at least the course of the disease could be predicted.

Kraepelin proposed two major groups of severe psychological disorders: dementia praecox (an early term for schizophrenia) and manic-depressive psychosis (an early term for bipolar disorder). He postulated a chemical imbalance as the cause of dementia praecox and an irregularity in metabolism as the explanation of manic-depressive psychosis. Though his theories about causes were not quite correct, Kraepelin's classification scheme nonetheless influenced the current diagnostic categories.

### Development of the WHO and DSM systems

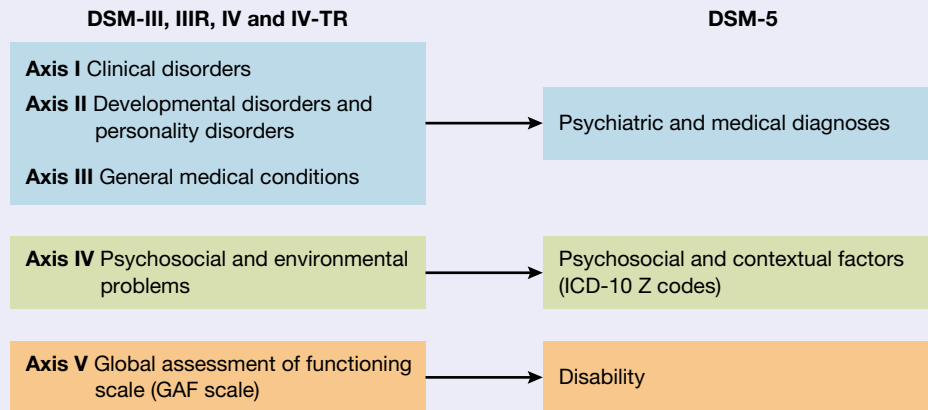
In 1939, the World Health Organization (WHO) added psychological disorders to the *International List of Causes of Death*. In 1948 the list was expanded to become the *International Statistical Classification of Diseases, Injuries and Causes of Death*, a comprehensive listing of all diseases, including a classification of psychological disorders. Unfortunately, the psychological disorders section was not widely accepted. Even though American psychiatrists had played a prominent role in the WHO effort, the American Psychiatric Association published its own *Diagnostic and Statistical Manual* (DSM) in 1952.

In 1969, the WHO published a new version, now referred to as the *International Classification of Disease* (ICD), which was more widely accepted. In the United Kingdom, a glossary of definitions was produced to accompany the WHO system. A second version of the American Psychiatric Association's DSM, DSM-II (1968), was similar to the WHO system. But true consensus still eluded the field. Even though DSM-II and the *British Glossary of Mental Disorders* specified some symptoms of diagnoses, the two systems defined different symptoms for a given disorder! Thus diagnostic practices still varied widely. A new version of the ICD, the ninth, was released in 1975. The next editions of the DSM, continuing with the present edition, have tried to achieve greater consensus with the ICD, which is currently in its tenth edition (the eleventh edition is expected in 2017).

In 1980, the American Psychiatric Association published an extensively revised diagnostic manual, DSM-III and a somewhat revised version, DSM-III-R, followed in 1987. One of the big changes in DSM-III that remained in place for 33 years was the introduction of the multiaxial system. Diagnoses were listed on separate dimensions or axes. As shown in figure 2.2, older versions of the DSM included five axes. This multiaxial classification system, by requiring judgements on each of the five axes, forced the diagnostician to consider a broad range of information. DSM-5 no longer uses these distinct axes.

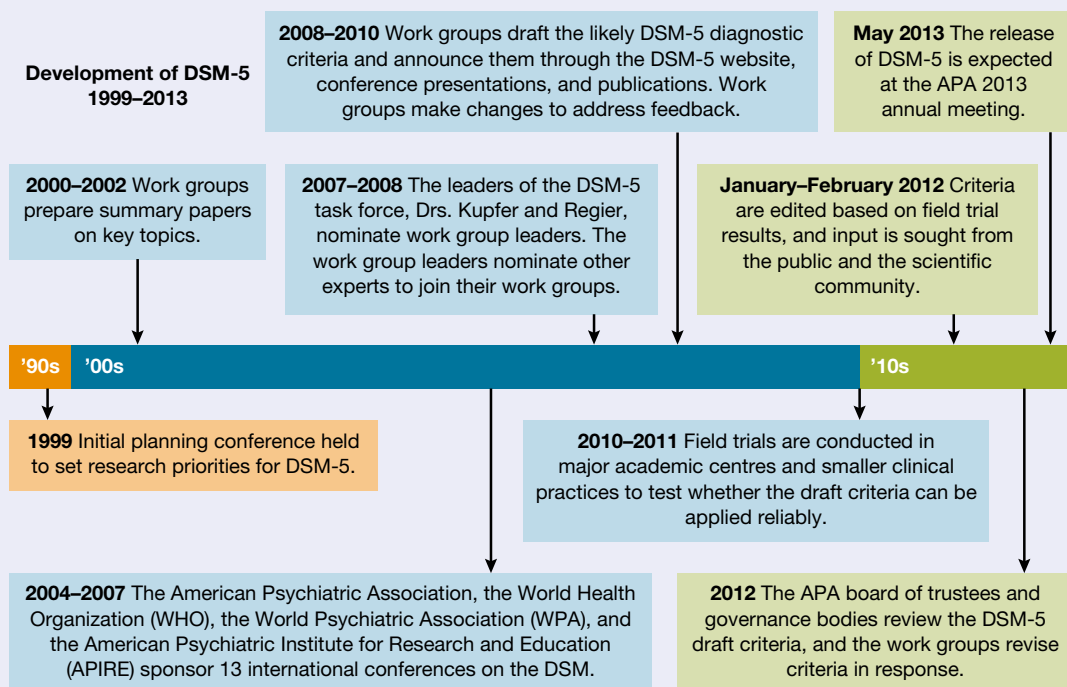
In 1988, the American Psychiatric Association began work on DSM-IV, which was published in 1994. Thirteen working groups, which included many psychologists, were established to critique DSM-III-R, review literature, analyse previously collected data and collect new data. Each work group tackled a different cluster of disorders. The committee adopted an important new approach — the reasons for changes in diagnoses would be explicitly stated and supported by data. In previous versions of the DSM, the reasons for diagnostic changes had not always been explicit. A revision to the supporting text of the DSM-IV, not the actual diagnostic criteria, was published in 2000 and named DSM-IV-TR, with the 'TR' standing for text revision.

**FIGURE 2.2** DSM-5 does not use the five axes of DSM-IV-TR



As shown in figure 2.3, the development of DSM-5 began in 1999. As with the process for DSM-IV, work groups reviewed each set of diagnoses. A series of study groups were also formed to consider issues that cut across diagnostic categories, such as life-span developmental approaches, gender and cross-cultural issues, general medical issues, impairment and disability, and diagnostic assessment instruments. These study groups conducted literature reviews and analyses and then provided feedback to the work groups regarding issues with specific diagnoses. As was done with DSM-IV, field trials were conducted on the new DSM-5 criteria in order to assess how they were performing in mental health settings. Additional revisions were made based on these data.

**FIGURE 2.3** Timeline for the development of DSM-5



In 2011, the **Australian Psychological Society (APS)** formed an 'Expert Reference Group' to coordinate a submission to the DSM-5 Task Force. According to the APS, most Australian clinicians and researchers use the DSM to inform their practice. The Expert Reference Group provided Australian psychologists with a vehicle to have input into the development of the DSM-5. Overall, the APS considered the DSM-5 an improvement on the previous revision and acknowledged that further work is needed to address the limitations of the classification system.

In 2014, the **New Zealand Psychological Society (NZPsS)** released a position statement on the DSM-5. The NZPsS expressed concern that the revisions lacked empirical support and clinical utility within the New Zealand health care system and cultural context. Instead, the NZPsS called for a 'paradigm shift' away from using a diagnostic system to employing a process of case conceptualisation. Case conceptualisation will be described later.

Although all changes to the DSM were to be based on research data, the DSM-5 Task Force emphasised that a high priority was to make the DSM-5 useful for clinicians. In the final stages of DSM-5 development, the leaders made changes that were not always consistent with the data and recommendations of the work groups (see the chapter on personality disorders for a stark example of this problem). To protect the process from commercial interests, all work group members signed conflict-of-interest agreements, stating that they would limit their income to \$10 000 or less per year from pharmaceutical and technology companies and similar industry groups. The crafters of the DSM-5 aimed to create a living document that will change as new research evidence emerges. New editions, then, are already on the horizon even as DSM-5 emerges and the DSM-5 website ([www.dsm5.org](http://www.dsm5.org)) continues to post such changes.

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### QUESTIONS

1. What are the two classification systems used for diagnosis?
2. How is the DSM used to inform the practice of psychology?

## Changes in DSM-5

The DSM-5 includes many changes from the last edition, DSM-IV-TR. Indeed, even conventions for labelling the edition have shifted — the Roman numerals used to denote the edition (e.g., DSM-IV) are replaced with Arabic numbers (i.e., DSM-5) to facilitate electronic printing. We will cover many of the changes as we discuss specific disorders in the chapters throughout this text. Here, then, we cover some of the major debates and changes that have implications across diagnoses.

### Removal of the multiaxial system

As shown in figure 2.2, the multiaxial system first developed for DSM-III in 1980 was removed in DSM-5. In place of the first three axes, clinicians are simply to note psychiatric and medical diagnoses. The codes for the Psychosocial and Environmental Problems Axis were changed to be more similar to those used by the international community in the World Health Organization's (WHO) International Classification of Diseases (ICD). Axis V is removed in DSM-5; instead, the World Health Organization Disability Assessment Schedule (WHODAS) is included in the DSM-5 appendix of assessment measures in need of further study.

### Organising diagnoses by causes

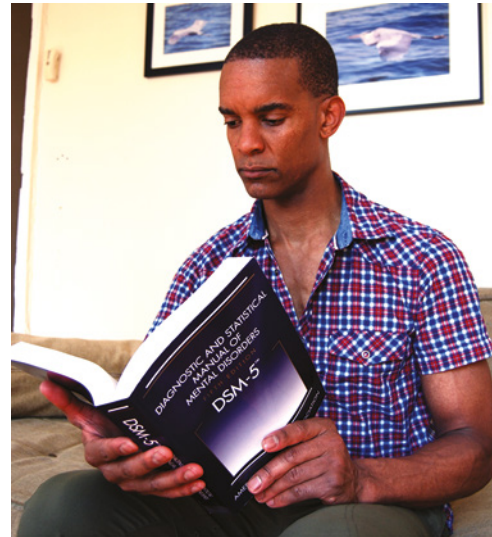
DSM-5 defines diagnoses on the basis of symptoms. Some have argued that advances in our understanding of aetiology (causes) could help us rethink this approach. For example, schizophrenia and schizotypal personality disorder share a great deal of genetic overlap. Could these ties be reflected in the diagnostic system? Others have proposed organising diagnoses based on parallels in neurotransmitter activity, temperament, emotion dysregulation or social triggers. After considerable review during the development of DSM-5, it became clear that our knowledge base is not yet strong enough to organise diagnoses around aetiology (Hyman, 2010). With the exception of IQ tests

to help with the diagnosis of intellectual disability (formerly known as mental retardation) or polysomnography for sleep disorders, we have no laboratory tests, neurobiological markers or genetic indicators to use in making diagnoses. The DSM-5 will continue to use symptoms as the basis for diagnosis.

On the other hand, changes were made to reflect growing knowledge of aetiology. The DSM-IV-TR diagnoses were clustered into chapters based on similarity of symptoms, but in the DSM-5, the chapters were reorganised to reflect patterns of comorbidity and shared aetiology (see figure 2.4). For example, in DSM-IV-TR, obsessive-compulsive disorder was included in the chapter on anxiety disorders because it contained anxiety symptoms. The aetiology of this disorder, though, seems to involve distinct genetic and neural influences compared to other anxiety disorders, as we discuss in a later chapter. To reflect this, the DSM-5 included a new chapter for obsessive-compulsive and other related disorders. This new chapter includes disorders that often co-occur and share some risk factors, including obsessive-compulsive disorder, hoarding disorder and body dysmorphic disorder.

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DSM-5 is the current diagnostic system of the American Psychiatric Association. It was released in 2013.



### **Enhanced sensitivity to the developmental nature of psychopathology**

In DSM-IV-TR, childhood diagnoses were considered in a separate chapter. Most of those diagnoses have now been moved into other relevant chapters of DSM-5, to highlight the continuity between childhood and adulthood forms of disorder. For example, children who experience separation anxiety may be at greater risk for developing anxiety disorders as adults. In DSM-5, separation anxiety disorder was moved to the chapter on anxiety disorders. Across diagnoses, more detail is provided about the expression of symptoms in younger populations.

### **New diagnoses**

Several new diagnoses were added to DSM-5. For example, disruptive mood dysregulation disorder was added to address the growing number of children and adolescents who are seen by clinicians due to severe mood changes and irritability as well as some of the symptoms of mania. Many of these youth do not meet the full criteria for mania (the defining feature of bipolar disorder) but were often incorrectly diagnosed with bipolar disorder because no other category seemed to fit their symptoms. It is hoped that by including this diagnosis, the overdiagnosis of bipolar disorder in children and adolescents will be lessened. Other new diagnoses include hoarding disorder, binge eating disorder, premenstrual dysphoric disorder and gambling disorder.

### **Combining diagnoses**

Some of the DSM-IV-TR diagnoses were combined because there was not enough evidence for differential aetiology, course or treatment response to justify separate diagnostic categories. For example, the DSM-IV-TR diagnoses of substance abuse and dependence were replaced with the DSM-5 diagnosis of substance use disorder. The DSM-IV-TR diagnoses of hypoactive sexual desire disorder and female sexual arousal disorder were replaced with the DSM-5 diagnosis of female sexual interest/arousal disorder. The DSM-IV-TR diagnoses of autism and Asperger's disorder were replaced with the DSM-5 diagnosis of autism spectrum disorder.

**FIGURE 2.4** Chapters in DSM-5

DSM-5 Chapters	
Neurodevelopmental disorders	
Elimination disorders	
Neurocognitive disorders	
Substance-related and other addictive disorders	
Schizophrenia spectrum and psychotic disorders	
Bipolar and related disorders	
Depressive disorders	
Anxiety disorders	
Obsessive-compulsive and related disorders	
Trauma- and stressor-related disorders	
Somatic symptom and related disorders	
Dissociative disorders	
Sexual dysfunctions	
Gender dysphoria	
Paraphilic disorders	
Feeding and eating disorders	
Sleep–wake disorders	
Disruptive, impulse control, and conduct disorders	
Personality disorders	
Other disorders	
Medication–movement disorders and other adverse effects of medication	
Other conditions that may be a focus of clinical attention	

## Ethnic and cultural considerations in diagnosis

Psychological disorders are universal. There is not a single culture in which people are free of psychological disorders. But there are many different cultural influences on the risk factors for psychological disorders (e.g., social cohesion, poverty, access to drugs of abuse and stress), the types of symptoms experienced, the willingness to seek help and the treatments available. Sometimes these differences

across cultures are profound. For example, it is estimated that there is only one psychiatrist for every 2 million people living in sub-Saharan Africa (World Health Organization, 2001). In Australia, just under 1.8 million people made use of mental health services in 2009–10 (Whiteford, Ferrari, Baxter, Charlson, & Degenhardt, 2014). The number of people seeking treatment for a mental health condition has increased in Australia from 37 percent in 2006–07 to 46 percent in 2009–10. This increase is thought to be due to the Australian government's Better Access Scheme, which gives Australian citizens government-subsidised access to mental health services (Whiteford et al., 2014).

In 2004–05, Indigenous Australians were twice as likely as non-Indigenous Australians to report high or very high levels of psychological distress (Australian Institute of Health and Welfare, 2010). This difference is thought to be due to disadvantage experienced by Indigenous Australians. Further, data from the Australian National Survey of Mental Health and Wellbeing found that first generation immigrants with non-English speaking backgrounds had lower evidenced prevalence of common mental health disorders in comparison to the Australian-born participants. This group difference is referred to as the 'healthy migrant hypothesis'. This group difference was not found when comparing Australian-born participants with second generation immigrant participants (Liddell, Nickerson, Sartor, Ivancic, & Bryant, 2016). Cultural differences do not always play out in the way one might expect. Unexpected cultural differences were also found in a major study conducted by the World Health Organization where outcomes for schizophrenia were more favourable in Nigeria, India and Colombia than in more industrialised countries, including the United States (Sartorius et al., 1986; Jablensky et al., 1992). As shown in table 2.2, rates of psychological disorders tend to be higher in New Zealand, Australia and the United States than in many other countries.

If we hope to understand how culture influences risk, symptom expression and outcomes, we need a diagnostic system that can be applied reliably and validly in different countries and cultures. To facilitate international communication, the DSM-5 includes a list that cross-references the DSM diagnoses with the International Statistical Classification of Diseases and Related Health Problems (ICD) codes.

Previous editions of the DSM were criticised for their lack of attention to cultural and ethnic variations in psychopathology. DSM-5 added features to enhance cultural sensitivity by including a section on culture-related diagnostic issues for nearly all disorders and an appendix devoted to developing a culturally informed case formulation. This appendix includes a cultural formulation interview consisting of 16 questions clinicians can use to help understand how culture may be shaping the clinical presentation. There is also a section on cultural concepts of distress that distinguishes syndromes that appear in particular cultures, culturally specific ways of expressing distress and cultural explanations about the causes of symptoms, illness and distress.

In the cultural formulation, clinicians are cautioned not to diagnose symptoms unless they are atypical and problematic within a person's culture. People vary in the degree to which they identify with their cultural or ethnic group. Some value assimilation into the majority culture, whereas others wish to maintain close ties to their cultural background. In general, clinicians are advised to be constantly mindful of how culture and ethnicity influence diagnosis and treatment. Attention needs to be paid to how culture can shape the symptoms and expression of a given disorder. For example, Malaysian Chinese are likely to report a somatic symptom, such as insomnia, as their presenting complaint when experiencing a major depressive episode, whereas Australian Caucasians are likely to describe affective and cognitive symptoms when experiencing depression (Parker, Cheah, & Roy, 2001).

In evaluating symptoms, clinicians also need to be aware that cultures may shape the language used to describe distress. In many cultures, for example, it is common to describe grief or anxiety in physical terms — 'I am sick in my heart' or 'My heart is heavy' — rather than in psychological terms.

**TABLE 2.2** Twelve-month prevalence rates of the most common psychological disorders by country

Country	Anxiety disorders	Mood disorders	Substance disorders	Any psychological disorder
Australia	6.2	14.4	5.1	20.0
New Zealand	24.9	20.2	12.3	39.5
Americas				
Colombia	10.0	6.8	2.8	17.8
Mexico	6.8	4.8	2.5	12.2
United States	18.2	9.6	3.8	26.4
Europe				
Belgium	6.9	6.2	1.2	12.0
France	12.0	8.5	0.7	18.4
Germany	6.2	3.6	1.1	9.1
Italy	5.8	3.8	0.1	8.2
Netherlands	8.8	6.9	3.0	14.9
Spain	5.9	4.9	0.3	9.2
Middle East and Africa				
Lebanon	11.2	6.6	1.3	16.9
Nigeria	3.3	0.8	0.8	4.7
Asia				
Japan	5.3	3.1	1.7	8.8
China	3.2	2.5	2.6	9.1

**Notes:**

- (a) Anxiety disorders include agoraphobia, generalised anxiety disorder, obsessive-compulsive disorder, panic disorder, post-traumatic stress disorder, social phobia and specific phobia. Mood disorders include bipolar I and II disorders, dysthymia and major depressive disorder. Substance disorders include alcohol or drug abuse or dependence. Values are percentages.
- (b) In the European countries, bipolar disorders and non-alcohol-related substance use disorders were not assessed. Obsessive-compulsive disorder was not assessed in Asian countries.

**Source:** Australian data from the 2007 National Survey of Mental Health and Wellbeing (Australian Institute of Health & Welfare, 2010); New Zealand data from the New Zealand Mental Health Survey, 2003–4 (Oakley Browne, Wells, & Scott, 2006); data for remaining countries from the WHO World Mental Health Survey Consortium (2004).

The DSM-5 includes nine cultural concepts of distress in the ‘Glossary of cultural concepts of distress’ to describe syndromes that are likely to be seen within specific regions. The following are some examples of syndromes listed in the DSM-5 glossary.

- *Dhat syndrome* is a term used in India to refer to severe anxiety about the discharge of semen.
- *Shenjing shuairuo* (neurasthenia) is a common diagnosis in China, a syndrome characterised by fatigue, dizziness, headaches, pain, poor concentration, sleep problems and memory loss.
- *Taijin kyofusho* is the fear that one could offend others through inappropriate eye contact, blushing, a perceived body deformation or one’s own foul body odour. This disorder is most common in Japan, but cases have been reported in the United States. Japanese cultural norms appear to prescribe more careful attention to social appropriateness and hierarchy, perhaps intensifying the risk of these symptoms (Fabrega, 2002).
- *Ataque de nervios* is intense anxiety and fear of screaming and shouting uncontrollably found among people from Latino/Latina cultures (e.g., Lewis-Fernandez et al., 2010).

Although not listed in the DSM-5 glossary, other culturally-relevant syndromes have been the focus of research. These include the following.

- *Amok* is a dissociative episode in which there is a period of brooding followed by a violent and sometimes homicidal outburst. The episode tends to be triggered by an insult and is found primarily among men. Persecutory delusions are often present as well. The term is Malaysian and is defined by the dictionary as a murderous frenzy. You may have heard the phrase ‘run amok’.

- *Ghost sickness* is an extreme preoccupation with death and those who have died, found among certain Native American tribes.
- *Hikikomori* (withdrawal) is a syndrome observed in Japan, Taiwan and South Korea in which an individual, most often an adolescent boy or young adult man, shuts himself into a room (e.g., bedroom) for a period of six months or more and does not socialise with anyone outside the room.

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The core symptoms of depression appear to be similar cross-culturally.



### CLINICAL CASE

#### **Amena — an example of diagnosis**

Amena was smuggled out of Afghanistan by her father when she was 14 years old. Amena's mother had died six months earlier. Amena and her father flew into Indonesia on false passports before coming to Australia on an asylum seeker boat. Amena and her father spent 18 months in a detention centre before being settled in Australia. Amena's father has had difficulty finding work and they currently live in a rough neighbourhood. Amena's English was fairly good when she came to Australia and she has picked up many of the nuances of the language since arriving in the country. For the past two years, she has been dating a boy in her school. They have been fairly constant companions and she describes him as the one person she would turn to if she was feeling upset. Amena seemed to rely on her boyfriend too much — she asked for his advice on both small and large decisions and she seemed wary of social interactions when he wasn't present. Her father disapproved of their relationship and his attempts to keep them apart only pushed them closer together.

With little warning, her boyfriend announced that he wanted to break up with her. Amena was extremely distressed by this change and reported that almost immediately she was unable to sleep or eat. She lost weight rapidly and found herself unable to concentrate on her schoolwork. Friends complained that she no longer wanted to talk during lunch or by phone. After two weeks of steadily feeling worse, Amena left a suicide note and disappeared. Police found her the next day in an abandoned home, holding a bottle of medication. She reported that she had been sitting there all night, considering ending her life. Amena's father reported that he had never seen her in such a distressed state but noted that a few other family members had struggled with periods of sadness. Still, these family members in Afghanistan had neither attempted suicide nor received any formal treatment. Instead, their sadness was viewed as a normal response to the difficulties they faced in their country. After the police found Amena, she was hospitalised for intensive treatment.

### DSM-5 diagnosis

Diagnoses: major depressive disorder, dependent personality disorder, post-traumatic stress disorder  
Important psychosocial/contextual factors: low income (ICD-10 Z59.6)

### QUESTIONS

1. How does Amena's cultural background and experience as an asylum seeker impact on diagnosis?
2. What would be the consequences of not considering Amena's cultural background and experience as an asylum seeker on diagnosis and subsequent treatment of Amena?

Some have argued that we should try to identify broad syndromes that can be identified across cultures and, in this light, have argued against differentiating cultural concepts of distress from other diagnostic syndromes (Lopez-Ibor, 2003). In support of this position, they point towards a number of cultural concepts of distress that are not so different from the main DSM diagnoses. For example, Suzuki and colleagues (2003) pointed out that the symptoms of *taijin kyofusho* overlap with those of social anxiety disorder (excessive fear of social interaction and evaluation) and body dysmorphic disorder (the mistaken belief that one is deformed or ugly), which are disorders described in the DSM-5. Other syndromes may reflect the common concerns of anxiety and distress, with the content shaped by life circumstances and values (Lopez-Ibor, 2003). Hence, some researchers believe it is important to look for commonalities across cultures.

In contrast, others believe that culturally relevant concepts of distress are central, because local and personal meanings are a key issue in understanding psychological disorders (Gaw, 2001). Still others have argued that many of the diagnostic categories from the DSM systems have been 'exported' across the world, often with little consideration of how culture impacts the symptom presentations, causes and treatment decisions (Watters, 2010). Whether one advocates for a cross-cultural or culture-specific approach to diagnosis, all mental health professionals ought to, at the very least, be aware of the cultural influences that can and do influence diagnostic practices (Sue, Yan Cheng, Saad, & Chu, 2012).

A therapist must be mindful of the role of cultural differences in the ways in which people describe their problems.



### RETURNING TO CLINICAL CASE

#### Roxanne: a second example of a diagnosis

Previously, we described the case of Roxanne, who was brought to the emergency department of her local hospital by the police. The DSM-5 and DSM-IV-TR diagnosis for Roxanne might look as follows.

### DSM-5 diagnosis

Diagnoses: bipolar I disorder, current or most recent episode manic; high blood pressure  
Psychosocial/contextual factors: problems with housing (homeless; ICD-10 Z59.0)

## Specific criticisms of the DSM

Some specific questions and concerns have been raised about the DSM. We review some of these concerns in the following sections.

### Too many diagnoses?

DSM-5 contains more than 300 different diagnoses. Some have criticised the burgeoning number of diagnostic categories (see table 2.3). As one example, the DSM-5 continues to include a category for acute stress disorder in order to capture symptoms in the first month after a severe trauma. This has led to some researchers questioning whether relatively common reactions to trauma should be pathologised by diagnosing them as a psychological disorder (Harvey & Bryant, 2002). Gambling disorder is added to DSM-5 under the chapter ‘Substance-related and other addictive disorders’. By expanding its coverage, the authors of the DSM seem to have made too many problems into psychological disorders without good justification for doing so.

**TABLE 2.3** Number of diagnostic categories per edition of DSM

Edition of DSM	Number of categories
DSM I	106
DSM-II	182
DSM-III	265
DSM-III-R	292
DSM-IV-TR	297
DSM-5	347

Others argue that the system includes too many minute distinctions based on small differences in symptoms. One side effect of the huge number of diagnostic categories is a phenomenon called **comorbidity**, which refers to the presence of a second diagnosis. Comorbidity is the norm rather than the exception. Among people who met criteria for at least one DSM-IV-TR psychiatric diagnosis, 45 percent meet criteria for at least one more psychiatric diagnosis (Kessler, Berglund, Demler et al., 2005). Some argue that this overlap is a sign that we are dividing syndromes too finely (Hyman, 2010).

A further problem with the large number of diagnoses is that many risk factors seem to trigger more than one disorder. For example, some genes are associated with an increase in the risk of externalising disorders (see the chapter on disorders of childhood) as a whole (Kendler, Hettema, Butera, Gardner, & Prescott, 2003). Early trauma, dysregulation of stress hormones, tendencies to attend to and remember negative information about the self and neuroticism all seem to increase risk for many anxiety disorders as well as mood disorders (Harvey, Watkins, Mansell, & Shafran, 2004). Anxiety and mood disorders also seem to share overlap in genetic risk (Kendler, Jacobson, Prescott, & Neale, 2003), diminished function of a brain region called the prefrontal cortex (Hyman, 2010) and low serotonin function (Carver, Johnson, & Joormann, 2008). Similarly, selective serotonin reuptake inhibitors (SSRIs), such as Prozac, often seem to relieve symptoms of anxiety as well as depression (Van Ameringen, Lane, Walker et al., 2001). Based on this information it could be concluded that different diagnoses do not seem to be distinct in their aetiology or treatment.

Does this mean that we should lump some of the disorders into one category? Beliefs about lumping versus splitting differ. Some think we should keep the finer distinctions, whereas others believe we should lump (Caspi, Houts, Belsky et al., 2014; Watson, 2005). Among people who think there are too many diagnostic categories, several researchers have considered ways to collapse disorders into broader categories. To begin with, some disorders seem to co-occur more frequently than others. Some have argued that childhood conduct disorder, adult antisocial personality disorder, alcohol use disorder and substance use disorder co-occur so often that they should be considered different manifestations of one

underlying disease process or vulnerability (Krueger, Markon, Patrick, & Iacono, 2005). These different types of problems could be jointly considered ‘externalising disorders’.

The authors of DSM-5 took modest steps towards addressing these concerns. First, in a few cases, two disorders were combined into one disorder. For example, as noted previously, the DSM-IV-TR diagnoses of substance abuse and dependence are replaced with the DSM-5 diagnosis of substance use disorder. The changes in DSM-5 are small, though. Comorbidity will remain the norm. Second, the authors created a cross-cutting symptom assessment measure to allow clinicians to assess for those symptoms that cut across the DSM-5 diagnostic boundaries, such as depressed or anxious mood, sleep problems and anger (Narrow et al., 2013).

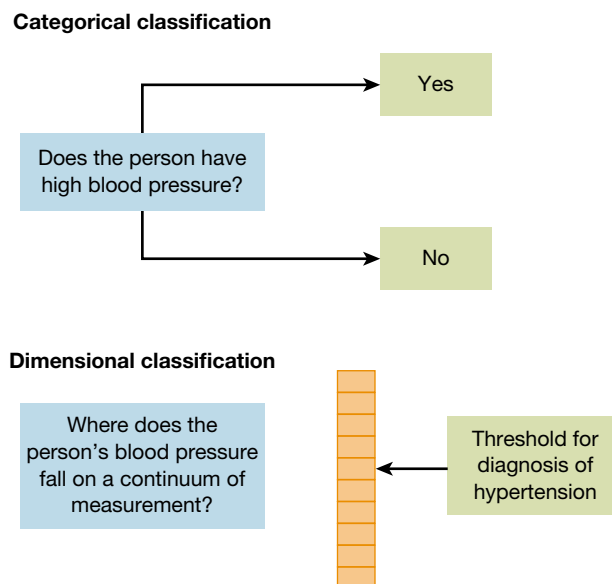
The National Institute of Mental Health is tackling this problem by working on a new diagnostic system that seeks to create (many fewer) categories based on common causes. Termed the **Research Domain Criteria**, or **RDoC**, this system is currently conceived as a roadmap for research that will lead to the development of a new classification system that is based on neuroscience and genetic data rather than just clinical symptoms (Insel, 2014).

### Categorical classification versus dimensional classification

In the DSM-5, clinical diagnoses are based on **categorical classification**. Does the person have schizophrenia or not? Do the symptoms fit the category of mania or not? For example, in table 2.1 we see that the diagnosis of mania requires the presence of three symptoms plus mania and excessive energy. But why require three symptoms rather than two or five? A categorical system forces clinicians to define one threshold as ‘diagnosable’. There is often little research support for the threshold defined. Categorical diagnoses foster a false impression of categories with actual, hard boundaries.

It may be more helpful to know the severity of symptoms as well as whether they are present. In contrast to categorical classification, **dimensional diagnostic systems** describe the *degree* of an entity that is present (e.g., a 1-to-10 scale of anxiety, where 1 represents minimal and 10, extreme). (See figure 2.5 for an illustration of the difference between dimensional and categorical approaches.)

**FIGURE 2.5** Categorical versus dimensional systems of diagnosis



One reason categorical systems are popular is that they define a threshold for treatment. Consider high blood pressure (hypertension). Blood pressure measurements form a continuum, which clearly fits a dimensional approach; yet by defining a threshold for high blood pressure, doctors can feel more certain

about when to offer treatment. Similarly, a threshold for clinical depression may help demarcate a point where treatment is recommended. Although the cut-offs are likely to be somewhat arbitrary, they can provide helpful guidance.

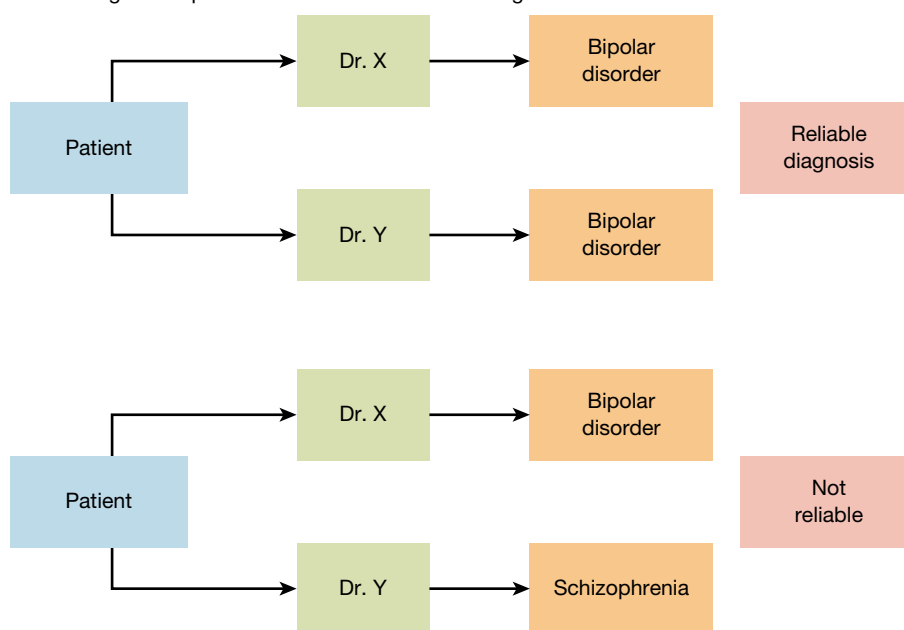
Despite some debate, particularly for personality disorders, DSM-5 preserved a categorical approach to diagnosis. A dimensional approach to personality disorders has been included in the appendix, but other diagnoses are based on categorical classification. DSM-5 did add severity ratings for nearly all disorders, however. Thus, DSM-5 is taking a first step towards including dimensions alongside the current categories.

The DSM-5 includes the category ‘unspecified’, which is to be used when a person meets many but not all of the criteria for a diagnosis (this was called ‘not otherwise specified’ in DSM-IV-TR). Unfortunately, far too many people will probably fit the ‘unspecified’ category.

## Reliability of the DSM in everyday practice

Suppose you were concerned about your mental health and you went to see two psychologists. Consider the distress you would feel if the two psychologists disagreed — one told you that you had schizophrenia and the other told you that you had bipolar disorder. Diagnostic systems must have high interrater reliability to be useful. Before DSM-III, reliability for DSM diagnoses was poor, mainly because the criteria for making a diagnosis were not clear (see figure 2.6 for an illustration of interrater reliability).

**FIGURE 2.6** Interrater reliability. In this example, the diagnosis of the first person is reliable — both clinicians diagnose bipolar disorder — whereas the diagnosis of the second is not reliable.



The increased explicitness of the DSM criteria has improved reliability for many diagnoses (see table 2.1). Nonetheless, because clinicians might not rely on the criteria precisely, the reliability of the DSM in everyday usage may be lower than that seen in research studies. Even when following criteria, there is some room for disagreement in DSM-5. For example, in the criteria for mania, mood is supposed to be ‘abnormally’ elevated. What is abnormal? Such judgements set the stage for the insertion of cultural biases as well as the clinician’s own personal ideas of what the average person should be doing. Because different clinicians may adopt different definitions for symptoms such as ‘abnormally elevated mood’, achieving high reliability can be a challenge.

Evidence from the DSM-5 field trials where the diagnostic criteria were tested at several different mental health treatment facilities suggests that the DSM still has work to do when it comes to reliability

(see table 2.4). Some have argued that expecting high reliability is unrealistic, particularly since the reliability of many medical diagnoses is not all that great either (Kraemer, Kupfer, Clarke, Narrow, & Regier, 2012). Although this may be true, it is disconcerting to think that mental health professionals may not agree on a particular person's diagnosis.

**TABLE 2.4** Reliability data from the DSM-5 field trials

Diagnosis	Pooled reliability estimate
Schizophrenia	.46
Bipolar I disorder	.56
Post-traumatic stress disorder	.67
Borderline personality disorder	.54
ADHD	.61
Autism spectrum disorder	.69

*Note:* Numbers are kappa statistics — the closer to 1.0, the better the reliability.

*Source:* Adapted from Regier et al. (2013).

## How valid are diagnostic categories?

The DSM diagnoses are based on a pattern of symptoms. A diagnosis of schizophrenia, then, does not have the same status as a diagnosis of, say, diabetes, for which we have laboratory tests.

One way of thinking about diagnosis is to ask whether the system helps organise different observations (see figure 2.1). Diagnoses have construct validity if they help make accurate predictions. What types of predictions should a good diagnostic category facilitate? One would hope that a diagnosis would inform us about related clinical characteristics and about functional impairments. The DSM specifies that impairment or distress must be present to meet criteria for a diagnosis, so perhaps it is not surprising that diagnoses are related to functional impairments such as missed days at work. In Australia, around 680 000 workdays per month are lost due to absence for mental health reasons. Over half of these absences were due to depressive disorders (Lim, Sanderson, & Andrews, 2000). Further, the largest proportion of disability support pension recipients in Australia are people with a diagnosed mental health condition (Matthewson, Langworthy, & Higgins, 2015). Beyond capturing the most common difficulties for a person with a diagnosis, one would hope that a diagnosis would inform us about what to expect next — the likely course of the disorder and response to different treatments. Perhaps most importantly, one would hope that the diagnosis relates to possible causes of the disorder, for example, a genetic predisposition or a biochemical imbalance. A diagnosis with strong construct validity should help predict a broad range of characteristics.

We have organised this text around the major DSM diagnostic categories because we believe that they do indeed possess some construct validity. Certain categories have less validity than others, however, and we will discuss these gaps in the validity of specific diagnostic categories in later chapters.

## General criticisms of diagnosing psychological disorders

Although we described some advantages of diagnosis in the beginning of this chapter, it is also clear that diagnoses can have negative effects. Consider how your life might be changed by receiving the diagnosis of schizophrenia. You might become worried that someone will recognise your disorder. Or you might fear the onset of another episode. You might worry about your ability to deal with new challenges. The fact that you have a diagnosis of a psychological disorder could have a stigmatising effect. Friends and loved ones might treat you differently and employment might be hard to find.

Without doubt, hearing a diagnosis can be difficult. Research shows that people with psychological disorders are widely viewed negatively, and they and their families often encounter stigma as a result. For example, an Australian survey found that over half of all respondents with a mental illness had experienced stigma at some time at work (SANE Australia, 2013). Many have raised concerns that a

diagnosis might contribute to stigma. Research, however, suggests that people tend to view behaviours more negatively than the category labels. Sometimes labels may actually relieve stigma by providing an explanation for the symptomatic behaviour (Lilienfeld, Lynn, Ruscio, & Beyerstein, 2010). For example, disclosing a diagnosis of a mental illness to employers can result in workers accessing support that enables them to perform their job better while managing symptoms (Matthewson et al., 2015). Of course, making a diagnosis is still a serious process that warrants sensitivity and privacy. But it may not be fair to presume that diagnostic labels are the major source of stigma.

Another concern is that when a diagnostic category is applied, we may lose sight of the uniqueness of that person. Because of this concern, the American Psychological Association recommends that people avoid using words like *schizophrenic* or *depressive* to describe people. After all, we do not call people with medical illnesses by their disease (e.g., you aren't likely to hear someone with cancer described as the *canceric*). Rather, psychologists are encouraged to use phrases such as *a person with schizophrenia*.

Even with more careful language, some maintain that diagnosis leads us to focus on disorders and, in doing so, to ignore important differences among people. Unfortunately, this criticism ignores a fundamental truth: it is human nature to categorise whenever we think about anything. Some would argue, then, that if we use categories anyway, it is best to develop the categories systematically. If one accepts this perspective, then the question is how well the current system does in grouping disorders.

### Diagnosis versus case conceptualisation

Case conceptualisation is a way of making sense of a patient's difficulties in the context of a theoretical framework. By doing this, the clinician brings together the patient's experience with theory and research. While diagnosis is viewed as a cornerstone of mental health practice, **case conceptualisation** is a way of collaboratively working with patients to personalise service.

Collaborative case conceptualisation can be a way of encouraging the patient to participate fully in the therapeutic process; it can help to make complex problems more manageable and guide the direction of treatment. Diagnosis can be seen as a process of determining what is wrong with a person whereas case conceptualisation is a process of further understanding why the person is experiencing their difficulties.

How a clinician approaches case conceptualisation will depend on the theory they apply to the case; however, a popular approach is to use the 'Five Ps' as described by Macneil, Hasty, Conus, and Berk (2012).

1. *Presenting problem*: a comprehensive description of the problems the client has come to therapy to address. This is based on the client's description of the problems during assessment.
2. *Predisposing factors*: any possible contributors to the problem. These may include biological, genetic, environmental and/or psychological factors as well as any critical incidents such as past trauma.
3. *Precipitating factors*: significant events preceding the onset of the disorder.
4. *Perpetuating factors*: any current difficulties that are maintaining the presenting problem.
5. *Protective factors*: any strengths or supports that may be moderating the impact of the presenting problem and may be used as positive resources in the therapeutic process.

Case conceptualisation has been criticised for having low interrater reliability. However, interrater reliability can be improved with training in case conceptualisation practices and clinical experience (Flinn, Braham, & Nair, 2014).

## 2.3 Psychological assessment

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**LEARNING OUTCOME 2.3** Describe the goals, strengths and weaknesses of psychological approaches to assessment.

To make a diagnosis, mental health professionals can use a variety of assessment measures and tools. Beyond helping to make a diagnosis, psychological assessment techniques are used in other important ways. For example, assessment methods are often used to identify appropriate therapeutic interventions. Furthermore, repeated assessments are very useful in monitoring the effects of treatment over time. Assessments are also fundamental to conducting research on the causes of disorder.

We will see that beyond the first interview a clinician has with a patient, many of the assessment techniques stem from the paradigms presented in chapter 1. Here we discuss clinical interviews; measures for assessing stress; personality tests, including objective and projective tests; intelligence tests; and behavioural and cognitive assessment techniques. Although we present these methods individually, a complete psychological assessment of a person will often involve combining several assessment techniques. The data from the various techniques complement each other and provide a more complete picture of the person. In short, there is no one best assessment measure. Rather, using multiple techniques and multiple sources of information will provide the best assessment.

## Clinical interviews

Most of us have probably been interviewed at one time or another, although the conversation may have been so informal that we did not regard it as an interview. Mental health professionals use both formal and structured as well as informal and less structured clinical interviews in psychopathological assessment.

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Mental health professionals use both formal and informal clinical interviews in psychopathological assessment.



### Characteristics of clinical interviews

One way in which a **clinical interview** is different from a casual conversation is the attention the interviewer pays to how the respondent answers questions — or does not answer them. For example, if a person is recounting marital conflicts, the clinician will generally be attentive to any emotion accompanying the comments. If the person does not seem upset about a difficult situation, the answers probably will be understood differently from how they would be interpreted if the person was crying or agitated while relating the story.

Carrying out good clinical interviews requires great skill. Clinicians, regardless of the paradigm adopted, recognise the importance of establishing rapport with a person who seeks their professional help. The interviewer must obtain the trust of the person; it is naive to assume that a person will easily reveal information to another, even to an authority figure with the title ‘Doctor’. Even a person who sincerely, perhaps desperately, wants to recount intensely personal problems to a professional may not be able to do so without help.

Most clinicians empathise with their clients in an effort to draw them out and to encourage them to elaborate on their concerns. An accurate summary statement demonstrating that the clinician heard and understood what their client was saying can help sustain the momentum of the conversation. By adopting a non-judgemental approach to the client's disclosures, the client will feel more comfortable revealing personal and painful information (Egan, 2013).

Interviews vary in the degree to which they are structured. In practice, most clinicians probably operate from only the vaguest outlines. Exactly how information is collected is left largely up to the particular interviewer and depends, too, on the responsiveness and responses of the interviewee. Through years of training and clinical experience, each clinician develops ways of asking questions that he or she is comfortable with and that seem to draw out the information that will be of maximum benefit to the person. Thus, to the extent that an interview is unstructured, the interviewer must rely on intuition and general experience. As a consequence, unstructured clinical interviews are less reliable than structured interviews; that is, two interviewers may reach different conclusions about the same person.

### Structured interviews

At times, mental health professionals need to collect standardised information, particularly for making diagnostic judgements based on the DSM. To meet that need, investigators use a **structured interview**, in which the questions are set out in a prescribed fashion for the interviewer. One example of a commonly used structured interview is the Structured Clinical Interview (SCID) (Spitzer, Gibbon, & Williams, 1996). The current revision of the SCID is generally formatted around the structure of the DSM-5. This is shown in figure 2.7.

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Structured interviews are widely used to make reliable diagnoses.



The SCID is a branching interview; that is, a person's response to one question determines the next question that is asked. It also contains detailed instructions to the interviewer concerning when and how to probe in detail and when to go on to questions about another diagnosis. Most symptoms are rated on a three-point scale of severity, with instructions in the interview schedule for directly translating the symptom ratings into diagnoses. The initial questions pertaining to obsessive-compulsive disorder (discussed in a later chapter) are presented in figure 2.7. The interviewer begins by asking about obsessions. If the responses elicit a rating of 1 (absent), the interviewer turns to questions about compulsions. If the person's responses again elicit a rating of 1, the interviewer is instructed to go to the questions for

post-traumatic stress disorder. On the other hand, if positive responses (rating of 2 or 3) are elicited about obsessive-compulsive disorder, the interviewer continues with further questions about that problem.

**FIGURE 2.7** Sample item from the SCID

SCID-5-CV

Obsessive-Compulsive Disorder

Page 73

## G. OBSESSIVE-COMPULSIVE DISORDER and POSTTRAUMATIC STRESS DISORDER

### Obsessive-Compulsive Disorder

	CURRENT OBSESSIVE-COMPULSIVE DISORDER	OBSESSIVE-COMPULSIVE DISORDER CRITERIA		
	In the past month, since (ONE MONTH AGO)...	A. Presence of obsessions, compulsions, or both: Obsessions are defined by (1) and (2):		
G1	<p>...have you been bothered by thoughts that kept coming back to you even when you didn't want them to, like being exposed to germs or dirt or needing everything to be lined up in a certain way? (What were they?)</p> <p>How about having urges to do something that kept coming back to you even though you didn't want them to, like an urge to harm a loved one? (What were they?)</p> <p>How about having images popping into your head that you didn't want, like violent or horrible scenes or something of sexual nature? (What were they?)</p> <p>IF YES TO ANY OF ABOVE: Have these (THOUGHTS/URGES/IMAGES) made you very anxious or upset?</p>	1. Recurrent and persistent thoughts, urges, or images that are experienced, at some time during the disturbance, as intrusive and unwanted, and that in most individuals cause marked anxiety or distress.	<p>↓ +</p> <p>Go to G3 (Compulsions), below.</p>	G1
G2	When you had these (THOUGHTS/URGES/IMAGES) did you try hard to get them out of your head? (What would you try to do?)	2. The individual attempts to ignore or suppress such thoughts, urges, or images, or to neutralize them with some other thought or action (i.e., by performing a compulsion).	<p>↓ +</p> <p>OBSESSIONS</p> <p>Go to G3 (Compulsions), below.</p>	G2
	In the past month, since (ONE MONTH AGO)...	Compulsions are defined by (1) and (2):		
G3	<p>...was there anything that you had to do over and over again and was hard to resist doing, like washing your hands again and again, repeating something over and over again until it "felt right," counting up to a certain number, or checking something many times to make sure that you'd done it right?</p> <p>Tell me about that. (What did you have to do?)</p>	1. Repetitive behaviors (e.g., hand washing, ordering, checking) or mental acts (e.g., praying, counting, repeating words silently) that the individual feels driven to perform in response to an obsession or according to rules that must be applied rigidly.	<p>↓ +</p> <p>Go to G5, next page.</p>	G3
G4	<p>IF UNCLEAR: Why did you have to do (COMPULSIVE ACT)? What would happen if you didn't do it?</p> <p>IF UNCLEAR: How many times would you do (COMPULSIVE ACT)? Are you doing (COMPULSIVE ACT)? more than really makes sense?</p>	2. The behaviors or mental acts are aimed at preventing or reducing anxiety or distress, or preventing some dreaded event or situation; however, these behaviors or mental acts either are not connected in a realistic way with what they are designed to neutralize or prevent, or are clearly excessive.	<p>↓ +</p> <p>COMPULSIONS</p> <p>Go to G5, next page.</p>	G4

Source: Reprinted by permission of New York State Psychiatric Institute Biometrics Research Division.

In practice, most clinicians review the DSM symptoms in an informal manner without using a structured interview. Note, however, that clinicians using unstructured diagnostic interviews tend to miss comorbid diagnoses that often accompany a primary diagnosis (Zimmerman & Mattia, 1999). When clinicians use an informal interview rather than a structured interview, the reliability of diagnoses also tends to be much lower (Garb, 2005). With adequate training, interrater reliability for structured interviews is generally quite good (Blanchard & Brown, 1998).

## Assessment of stress

Given its centrality to nearly all the disorders we consider in this text, measuring stress is clearly important in the total assessment picture. To understand the role of stress, we must first be able to define and measure it. Neither task is simple, as stress has been defined in many ways. See focus on discovery 2.2 for influential antecedents to our current conceptualisations of stress. Broadly, **stress** can be conceptualised as the subjective experience of distress in response to perceived environmental problems. Life stressors can be defined as the environmental problems that trigger the subjective sense of stress. Various scales and methods have been developed to measure life stress. Here we examine the most comprehensive measure of life stress: the Life Events and Difficulties Schedule (LEDS) as well as self-report checklist measures of stress.

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Stress can include major life events or daily hassles such as traffic jams.



### FOCUS ON DISCOVERY 2.2

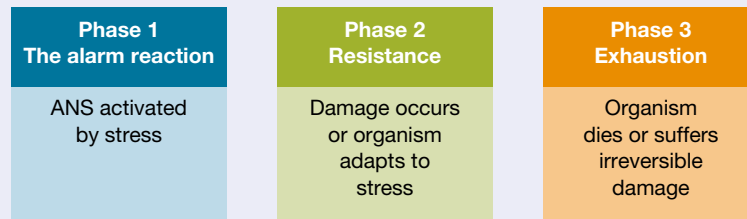
#### A brief history of stress

The pioneering work by the physician Hans Selye set the stage for our current conceptualisations of stress. He introduced the term *general adaptation syndrome* (GAS) to describe the biological response to sustained and high levels of stress (see figure 2.8). In Selye's model there are three phases of the response.

1. During the first phase, the alarm reaction, the autonomic nervous system is activated by the stress.
2. During the second phase, resistance, the organism tries to adapt to the stress through available coping mechanisms.
3. If the stressor persists or the organism is unable to adapt effectively, the third phase, exhaustion, follows and the organism dies or suffers irreversible damage (Selye, 1950).

In Selye's syndrome, the emphasis was on the body's response, not the environmental events that trigger that response. Psychological researchers later broadened Selye's concept to account for the diverse stress responses that people exhibited, including emotional upset, deterioration of performance or physiological changes such as increases in the levels of certain hormones. The problem with these response-focused definitions of stress is that the criteria are not clear-cut. Physiological changes in the body can occur in response to a number of things that we would not consider stressful (e.g., anticipating a pleasurable event).

**FIGURE 2.8** Selye's general adaptation syndrome



Other researchers defined stress as a stimulus, often referred to as a stressor, rather than a response and identified stress with a long list of environmental conditions, such as electric shock, boredom, catastrophic life events, daily hassles and sleep deprivation. Stimuli that are considered stressors can be major (the death of a loved one), minor (daily hassles, such as being stuck in traffic), acute (failing an exam) or chronic (a persistently unpleasant work environment). For the most part, they are experiences that people regard as unpleasant, but they can also be pleasant events.

Like response-based definitions of stress, stimulus-based definitions present problems. It is important to acknowledge that people vary widely in how they respond to life's challenges. A given event does not elicit the same amount of stress in everyone. For example, a family that has lost its home in a flood but has money enough to rebuild and strong social support from a network of friends nearby will experience less stress from this event than a family that has neither adequate money to rebuild nor a network of friends to provide social support.

Current conceptualisations of stress emphasise that how we perceive, or *appraise*, the environment determines whether a stressor is present. Stress is perhaps most completely conceptualised as the subjective experience of distress in response to perceived environmental problems. A final exam that is merely challenging to some students may be highly stressful to others who do not feel prepared to take it (whether or not their concerns are realistic).

## QUESTIONS

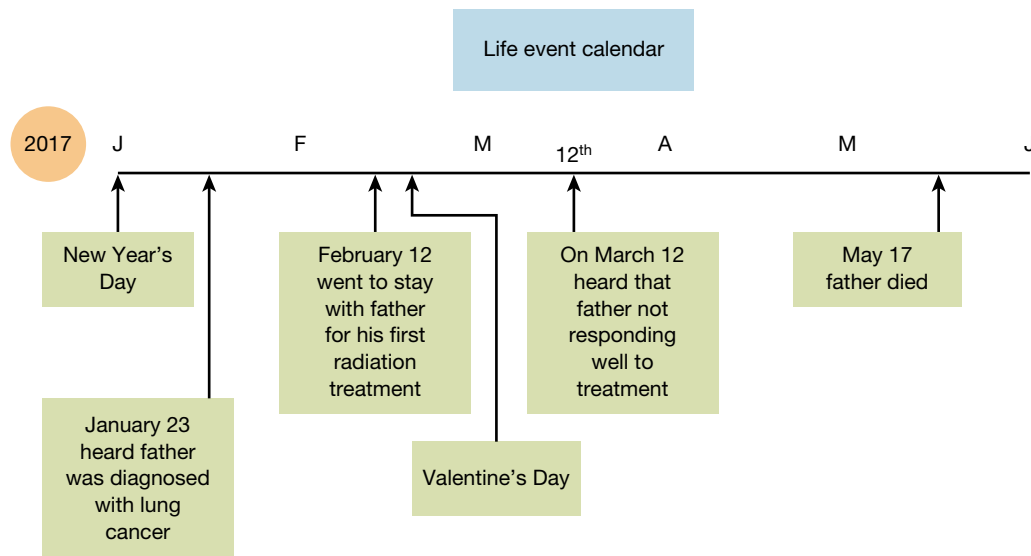
1. What are the limitations of response-based and stimulus-based definitions of stress?
2. Is stress always a negative thing? What are some positive aspects of stress?

## The Bedford College Life Events and Difficulties Schedule

This assessment is widely used to study life stressors (Brown & Harris, 1978). The LEDS includes an interview that covers over 200 different kinds of stressors. Because the interview is only semistructured, the interviewer can tailor questions to cover stressors that might only occur to a small number of people. The interviewer and the interviewee work collaboratively to produce a calendar of each of the major events within a given time period (see figure 2.9 for an example). After the interview, raters evaluate the severity and several other dimensions of each stressor. The LEDS was designed to address a number of problems in life stress assessment, including the need to evaluate the importance of any given life event in the context of a person's life circumstances. For example, pregnancy might have quite a different meaning for an unmarried 14-year-old girl compared to a 38-year-old woman who has been trying to

conceive for a long time. A second goal of the LEDS is to exclude life events that might just be consequences of symptoms. For example, if a person misses work because he or she is too depressed to get out of bed, any consequent job problems should really be seen as symptoms of the disorder rather than a triggering life event. Finally, the LEDS includes a set of strategies to carefully date when a life stressor occurred. Using this more careful assessment method, researchers have found that life stressors are robust predictors of episodes of anxiety, depression, schizophrenia and even the common cold (Brown & Harris, 1989; Cohen, Frank, Doyle et al., 1998).

**FIGURE 2.9** Example of a life events timeline. The LEDS interview is designed to capture the major stressors a person has encountered in the past year.



### Self-report stress checklists

Because intensive interview measures like the LEDS are so comprehensive, they take a good deal of time to administer. Often clinicians and researchers want a quicker way to assess stress and thus may turn to self-report checklists, such as the List of Threatening Experiences (LTE; Brugha & Cragg, 1990); the Psychiatric Epidemiological Research Interview Life Events Scale (PERL; Dohrenwend, Krasnoff, Askenasy, & Dohrenwend, 1978); or Perceived Stress Scale (PSS; Cohen, Kamarck, & Mermelstein, 1983). These checklists typically list different life events (e.g., death of a spouse, serious physical illness, major financial crisis) and participants are asked to indicate whether or not these events happened to them in a specified period of time. One difficulty associated with these types of measures is that there is a great deal of variability in how people view these events (Dohrenwend, 2006). For example, the death of a spouse could be the most horrible event ever for someone in a loving relationship. However, for someone in an abusive relationship, it might be the source of relief rather than stress. Other problems with such self-report checklists include difficulties with recall (Dohrenwend, 2006). For example, people may forget about some events. There is also evidence that people who are feeling depressed or anxious when they complete the measure may be biased in their responses. Perhaps because of these various issues influencing recall, test-retest reliability for life stress checklists can be low (McQuaid, Monroe, Roberts, & Johnson et al., 1992).

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The LEDS focuses on major stressors, such as deaths, job losses and romantic breakups.



## Personality tests

**Psychological tests** further structure the process of assessment. The two most common types of psychological tests are personality tests and intelligence tests. Here we will examine the two types of personality tests: self-report personality inventories and projective personality tests.

### Self-report personality inventories

In a **personality inventory**, the person is asked to complete a self-report questionnaire indicating whether statements assessing habitual tendencies apply to him or her. When these tests are developed, they are typically administered to many people to analyse how certain kinds of people tend to respond. Statistical norms for the test can thereby be established. This process is called **standardisation**. The responses of a particular person can then be compared with the statistical norms.

Perhaps the best known of these tests is the **Minnesota Multiphasic Personality Inventory (MMPI)**, developed in the early 1940s by Hathaway and McKinley (1943) and revised in 1989 (Butcher, Dahlstrom, Graham, Tellegen, & Kraemer, 1989). The MMPI is called multiphasic because it was designed to detect a number of psychological problems. Hundreds of items were tested in very large samples of people with and without a diagnosis. Sets of these items were established as scales. If a person answered many of the items in a scale in the same way as had people from a certain diagnostic group, his or her behaviour was expected to resemble that of the particular diagnostic group. These 10 scales are described in table 2.5.

**TABLE 2.5** Typical clinical interpretations of items similar to those on the MMPI-2

Scale		Sample item	Interpretation
	(Cannot say)	This is merely the number of items left unanswered or marked both true and false.	A high score indicates evasiveness, reading difficulties or other problems that could invalidate results of the test. A very high score could also suggest severe depression or obsessional tendencies.
L	(Lie)	I approve of every person I meet. (True)	Person is trying to look good, to present self as someone with an ideal personality.
F	(Infrequency)	Everything tastes sweet. (True)	Person is trying to look abnormal, perhaps to ensure getting special attention from the clinician.
K	(Correction)	Things couldn't be going any better for me. (True)	Person is guarded, defensive in taking the test, wishes to avoid appearing incompetent or poorly adjusted.
1.	Hs (Hypochondriasis)	I am seldom aware of tingling feelings in my body. (False)	Person is overly sensitive to and concerned about bodily sensations as signs of possible physical illness.
2.	D (Depression)	Life usually feels worthwhile to me. (False)	Person is discouraged, pessimistic, sad, self-deprecating, feeling inadequate.
3.	Hy (Hysteria)	My muscles often twitch for no apparent reason. (True)	Person has somatic complaints unlikely to be due to physical problems; also tends to be demanding and histrionic.
4.	Pd (Psychopathy)	I don't care about what people think of me. (True)	Person expresses little concern for social mores, is irresponsible, has only superficial relationships.
5.	Mf (Masculinity–Femininity)	I like taking care of plants and flowers. (True, female)	Person shows non-traditional gender characteristics (e.g., men with high scores tend to be artistic and sensitive).
6.	Pa (Paranoia)	If they were not afraid of being caught, most people would lie and cheat. (True)	Person tends to misinterpret the motives of others, is suspicious and jealous, vengeful and brooding.
7.	Pt (Psychasthenia)	I am not as competent as most other people I know. (True)	Person is overanxious, full of self-doubts, moralistic and generally obsessive-compulsive.
8.	Sc (Schizophrenia)	I sometimes smell things others don't sense. (True)	Person has bizarre sensory experiences and beliefs, is socially reclusive.
9.	Ma (Hypomania)	Sometimes I have a strong impulse to do something that others will find appalling. (True)	Person has overly ambitious aspirations and can be hyperactive, impatient and irritable.
10.	Si (Social introversion)	Rather than spend time alone, I prefer to be around other people. (False)	Person is very modest and shy, preferring solitary activities.

**Note:** The first four scales assess the validity of the test; there are seven other scales and methods for assessing validity not shown here. The numbered scales are the clinical or content scales. The Restructured Clinical (RC) Scales are not shown here.

**Sources:** Hathaway & McKinley (1943); revised by Butcher et al. (1989).

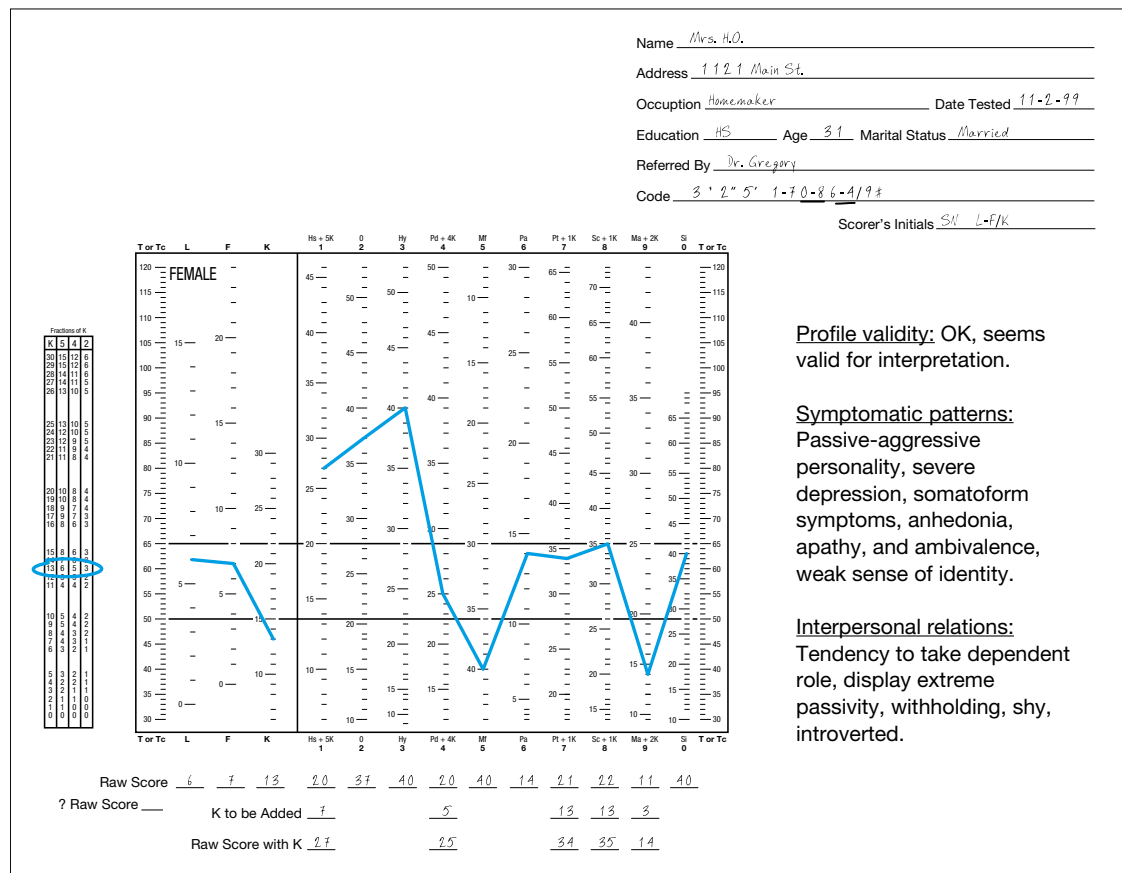
The MMPI-2 (Butcher et al., 1989) was designed to improve validity and acceptability. The original sample from the MMPI was composed mainly of white people from Minnesota and lacked representation of ethnic minorities. The 1989 version was standardised using a sample that was much larger and more representative of 1980 US Census figures. Several items containing allusions to sexual adjustment, bowel and bladder functions and excessive religiosity were removed because they were judged in some testing contexts to be needlessly intrusive and objectionable. Sexist wording was eliminated, along with outmoded idioms. New scales deal with substance abuse, emotions and marital problems.

The MMPI-2 was subsequently revised and updated in 2001, 2003 and 2009. In these revisions, new profiles with revised clinical scales were added. An extensive research literature shows that the MMPI-2 is reliable and has adequate criterion validity when it is related to diagnoses made by clinicians and to ratings made by spouses (Graham, 2011).

Like many other personality inventories, the MMPI-2 is typically administered and scored by computer. Figure 2.10 shows a hypothetical profile. Such profiles can be used in conjunction with other assessment measures to help with diagnosis, assessment of personality functioning and coping style, and identification of potential obstacles to treatment.

You may wonder whether it would be easy to fake answers that suggest no psychopathology. For example, a superficial knowledge of contemporary psychopathology research could alert someone that to be regarded as psychologically healthy, he or she must not admit to worrying a great deal about receiving messages from television.

**FIGURE 2.10** Hypothetical MMPI-2 profile



As shown in table 2.5, the MMPI-2 includes several ‘validity scales’ designed to detect deliberately faked responses. For example, an item on the lie scale might be, ‘I read the newspaper editorials every day’. The assumption is that few people would be able to endorse such a statement honestly. Persons who endorse a large number of the statements in the lie scale might be attempting to present themselves in a good light. High scores on the infrequency (F) scale also discriminate between people trying to fake psychopathology and people who actually have a psychological disorder (Bagby, Nicholson, Bacchionchi et al., 2002). If a person obtains high scores on the lie or infrequency (F) scale, his or her profile might be viewed with scepticism. People who are aware of these validity scales, however, can still effectively fake their profile (Baer & Sekirnjak, 1997; Walters & Clopton, 2000). In most testing circumstances, however, people do not want to falsify their responses because they want to be helped. Focus on discovery 2.3 discusses other issues surrounding the validity of self-report questionnaires.

### FOCUS ON DISCOVERY 2.3

#### Underreporting of stigmatised behaviours

Studies of self-reported drug use, sexual behaviour and violence on questionnaires or in face-to-face interviews highlight the importance of the setting in establishing the validity of what people will tell about their actions and attitudes (Ghanem, Hutton, Zenilman, Zimba, & Erbeling, 2005; Turner, Ku, Rogers, Lindberg, & Pleck, 1998).

In one study (Turner et al., 1998), findings from self-report questionnaires were compared with results from a novel self-report method — boys and young men (ages 15 to 19) listened by themselves through headphones to questions probing risky, often stigmatised behavioural practices and then indicated whether they had engaged in those behaviours by pressing keys on a computer keyboard labelled Yes and No. Compared to a matched control group who responded to the same items on a paper-and-pencil questionnaire, many more of the computer respondents admitted to having engaged in a range of high-risk behaviours. For example, they were almost 14 times more likely to report having had sex with an intravenous drug user (2.8 percent versus 0.2 percent), more than twice as likely to report having been paid for sex (3.8 percent versus 1.6 percent) and almost twice as likely to report having used cocaine (6.0 percent versus 3.3 percent). (One can safely assume that the differences would have been even greater if the boys had been interviewed by an adult researcher facing them across a table, another method that has been used to collect such survey data.) No significant differences showed up on questions directed at non-stigmatised or legal behaviours such as having had sex with a female in the preceding year (47.8 percent for computer users versus 49.6 percent for paper-and-pencil questionnaires) or having drunk alcohol in the past year (69.2 percent versus 65.9 percent).

In another study (Ghanem et al., 2005), men attending a clinic for sexually transmitted diseases treatment were randomly assigned to a face-to-face interview or an audio computer-assisted self interview (ACASI) when interviewed about high-risk sexual practices. Compared to the men who participated in the face-to-face interviews, those who participated in the ACASI were more likely to disclose sensitive information, such as same-sex sexual contact, number of sex partners in the past month and receptive anal sexual contact. Interestingly, though, men in both conditions were equally likely to disclose illicit drug use behaviours.

If these findings show nothing else, they strongly suggest that the frequencies of sensitive behaviour as determined by questionnaire or interview studies may be underestimated and that behaviours such as needle sharing and unsafe sex may be considerably more common than most people believe.

In Australia, participation in research is voluntary. Any disclosures made by participants in research must be kept confidential and information must be maintained in accordance with privacy laws. To ensure this happens, researchers must adhere to the *National Statement on Ethical Conduct in Human Research* (National Statement; NHMRC, 2007). The *National Statement* is a series of guidelines that researchers conducting research in Australia must abide by. Research in Australia involving human participants must be approved and monitored by a **Human Research Ethics Committee (HREC)**. Similarly, the **Health Research Council (HRC)** in New Zealand ensures that all research involving human participants meets ethical and legal standards. Research conducted in New Zealand must be approved and monitored by an HRC-approved ethics committee.

Further, in Australia, registered psychologists are bound by the **Australian Psychological Society's Code of Ethics** (the Code; APS, 2007) and the *Health Practitioner Regulation National Law Act 2009/2010* (the National Law) relevant to the state or territory in which they practise. According to the Code and National Law, psychologists must maintain the confidentiality of their patients unless breaching confidentiality is necessary for the safety of the public or of the patient. This is similarly the case in New Zealand where the conduct of psychologists is monitored by the New Zealand Psychologists' Board. It is thought that the promise of confidentiality will encourage patients to accurately report sensitive information.

### QUESTIONS

1. Why would people be more willing to disclose sensitive personal information on computer surveys than in face-to-face interviews despite the promise of confidentiality?
2. How do codes of conduct and ethical practice guidelines help to increase the validity of disclosures made by patients and research participants?

## Projective personality tests

A **projective test** is a psychological assessment tool in which a set of standard stimuli — inkblots or drawings — ambiguous enough to allow variation in responses is presented to the person. The assumption is that because the stimulus materials are unstructured and ambiguous, the person's responses will be determined primarily by unconscious processes and will reveal his or her true attitudes, motivations and modes of behaviour. This notion is referred to as the **projective hypothesis**.

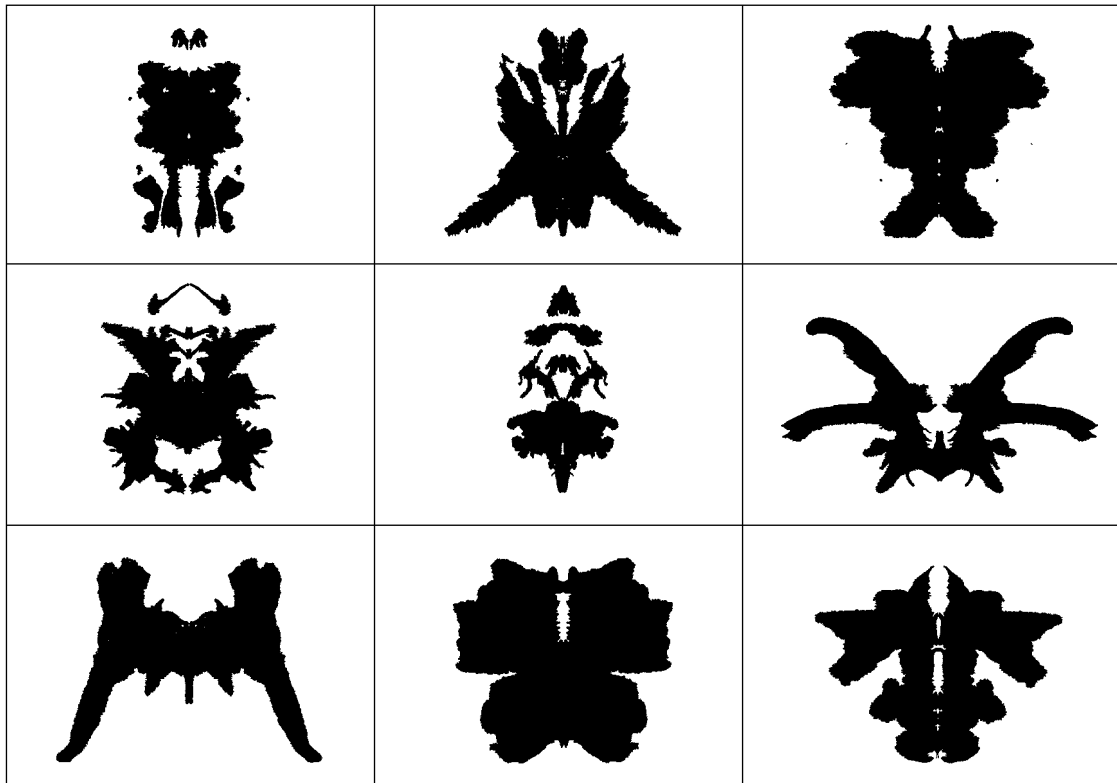
If a person reports seeing eyes in an ambiguous inkblot, for example, the projective hypothesis might be that the person tends towards paranoia. The use of projective tests assumes that the respondent would be either unable or unwilling to express his or her true feelings if asked directly. As you might have guessed, projective techniques are derived from the work of Freud and his followers (see chapter 1).

The **Thematic Apperception Test (TAT)** is a projective test. In this test a person is shown a series of black-and-white pictures one-by-one and asked to tell a story related to each. For example, a person seeing a picture of a boy observing a youth baseball game from behind a fence may tell a story that contains angry references to the boy's parents. The clinician may, through the projective hypothesis, infer that the person harbours resentment towards his or her parents. There are few reliable scoring methods for this test and the norms are based on a small and limited sample (i.e., few norms for people of different ethnic or cultural backgrounds). The construct validity of the TAT is also limited (Lilienfeld, Wood, & Garb, 2000). The **Rorschach Inkblot Test** is perhaps the best-known projective technique. In the Rorschach test, a person is shown 10 inkblots (for similar inkblots, see figure 2.11), one at a time and asked to tell what the blots look like. Half the inkblots are in black, white and shades of grey; two also have red splotches; and three are in pastel colours.

Exner (1978) designed the most commonly used system for scoring the Rorschach test. The Exner scoring system concentrates on the perceptual and cognitive patterns in a person's responses. The person's responses are viewed as a sample of how he or she perceptually and cognitively organises real-life situations (Exner, 1986). For example, Erdberg and Exner (1984) concluded from the research literature that respondents who express a great deal of human movement in their Rorschach responses (e.g., 'The man is running to catch a plane') tend to use inner resources when coping with their needs, whereas those whose Rorschach responses involve colour ('The red spot is a kidney') are more likely to seek interaction with the environment. (Rorschach suggested this approach to scoring in his original manual, *Psychodiagnostics: A Diagnostic Test Based on Perception* [1921], but he died only eight months after publishing his 10 inkblots and his immediate followers devised other methods of interpreting the test.)

**FIGURE 2.11**

In the Rorschach test, the person is shown a series of inkblots and is asked what the blots look like.



The Exner scoring system has norms, although the sample on which they are based was rather small and did not represent different ethnicities and cultures well. Regarding its reliability and validity, this work has enthusiastic supporters as well as equally vocal critics (Hunsley & Bailey, 1999; Lilienfeld et al., 2000; Meyer & Archer, 2001). Perhaps trying to make a blanket statement about the validity of the Rorschach (or the MMPI-2) is not the right approach. The test appears to have better validity in assessing some issues more than others. For example, limited evidence suggests that the Rorschach may have validity in identifying schizophrenia, borderline personality disorder and dependent personality traits, but it remains unclear whether it does so better than other assessment techniques (Lilienfeld et al., 2000). In other words, it is unclear whether the Rorschach provides information that could not be obtained more simply — for example, through an interview.

## Intelligence tests

Alfred Binet, a French psychologist, originally constructed tests to help the Parisian school board predict which children were in need of special schooling. Intelligence testing has since developed into one of the largest psychological industries. An **intelligence test**, often referred to as an IQ test, is a way of assessing a person's current mental ability. IQ tests are based on the assumption that a detailed sample of a person's current intellectual functioning can predict how well he or she will perform in school and most such tests are individually administered. The most commonly administered tests include the Wechsler Adult Intelligence Scale, 4th edition (WAIS-IV, 2008); the Wechsler Intelligence Scale for Children, 5th edition (WISC-IV, 2014); the Wechsler Preschool and Primary Scale of Intelligence,

4th edition (WPPSI-IV, 2012); and the Stanford–Binet, 5th edition (SB5, 2003); IQ tests are regularly updated and, like personality inventories, they are standardised.

Beyond predicting school performance, intelligence tests are also used in other ways:

- in conjunction with achievement tests, to diagnose learning disorders and to identify areas of strengths and weaknesses for academic planning
- to help assess whether a person has intellectual impairment
- to identify intellectually gifted children so that appropriate instruction can be provided in school
- as part of neuropsychological evaluations; for example, periodically testing a person believed to be suffering from dementia so that deterioration of mental ability can be followed over time.

IQ tests tap several functions believed to constitute intelligence, including language skills, abstract thinking, nonverbal reasoning, visual-spatial skills, attention and concentration and speed of processing. Scores on most IQ tests are standardised so that 100 is the mean (i.e., the average score) and 15 or 16 is the standard deviation (a measure of how scores are dispersed above and below the average). Approximately 65 percent of the population receives scores between 85 and 115. Approximately 2.5 percent of the population falls below 70 or above 130 (i.e., 2 standard deviations below or above the mean score of 100).

IQ tests are highly reliable (Canivez & Watkins, 1998) and have good criterion validity. For example, they distinguish between people who are intellectually gifted and those with intellectual impairment and between people with different occupations or educational attainment (Reynolds, Chastain, Kaufman, & McLean, 1997). They also predict educational attainment and occupational success (Hanson, Hunsley, & Parker, 1988), at least among Caucasians (see below for a discussion of cultural bias in assessment). Although IQ and educational attainment are positively correlated, what remains less clear is whether more education causes an increase in IQ or whether IQ causes one to attain more education (Deary & Johnson, 2010). Furthermore, although correlations between IQ scores and school performance are statistically significant, IQ tests explain only a small part of school performance; much more is unexplained by IQ test scores than is explained.

Of interest to the subject matter of this text, IQ is also correlated with health. In one study of over one million Scandinavian men, lower IQ scores at age 20 were associated with a greater risk of hospitalisation for schizophrenia, mood disorders or substance dependence some 20 years later, even after controlling for other possible contributing factors, such as the participants' families' socioeconomic status (Gale, Batty, Tynelius, Deary, & Rasmussen, 2010). A meta-analysis of 16 prospective, longitudinal studies found that lower IQ scores in early adulthood were associated with greater mortality risk (i.e., death) later in life, even after controlling for other variables such as socioeconomic status and educational attainment (Calvin et al., 2010).

Regarding construct validity, it is important to keep in mind that IQ tests measure only what psychologists consider intelligence. Factors other than what we think of as intelligence, however, also play an important role in how people will do in school, such as family and circumstances, motivation to do well, expectations, performance anxiety and difficulty of the curriculum. Another factor relevant to IQ test performance is called stereotype threat. It suggests that the social stigma of poor intellectual performance borne by some groups (e.g., women perform more poorly than men on mathematics tests) actually interferes with their performance on these tests. In one study demonstrating this phenomenon, groups of men and women were given a difficult mathematics test. In one condition, the participants were told that men scored higher than women on the test they were going to take (stereotype threat condition), while in the other condition they were told there were no gender differences in performance on the test. Only when the test was described as yielding gender differences did the women perform more poorly than the men (Spencer, Steele, & Quinn, 1999).

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IQ tests have many subtests, including this test to assess form perception and spatial ability.



Unfortunately, awareness of these stereotypes develops early. For example, a study revealed that children develop awareness of stereotypes regarding ethnicity and ability between the ages of 6 and 10, with 93 percent of children being aware of such stereotypes by age 10 (McKown & Weinstein, 2003). This awareness seems to influence stereotype threat (and performance). In the McKown and Weinstein (2003) study, children were asked to complete a puzzle task. Half of the children received instructions that the task reflected their ability (stereotype threat condition) and half the children received instructions that the test did not reflect their ability. African-American children who were aware of the stereotype about ethnicity and ability showed evidence of stereotype threat. Specifically, among African-American children, those who received the ability instructions performed more poorly on the puzzle task than the children who did not, suggesting that the instructions activated the stereotype and thus influenced their performance.

## Behavioural and cognitive assessment

Thus far, we have discussed assessment methods that measure personality traits and intellectual ability. Other types of assessment focus on behavioural and cognitive characteristics, including the following:

- aspects of the environment that might contribute to symptoms (e.g., an office location next to a noisy hallway might contribute to concentration problems)
- characteristics of the person (e.g., a person's fatigue may be caused in part by a cognitive tendency towards self-deprecation manifested in such statements as 'I never do anything right, so what's the point in trying?')

- the frequency and form of problematic behaviours (e.g., procrastination taking the form of missing important deadlines)
- consequences of problem behaviours (e.g., when a person avoids a feared situation, his or her partner offers sympathy and excuses, thereby unwittingly keeping the person from facing up to his or her fears).

The information necessary for a behavioural or cognitive assessment is gathered by several methods, including direct observation of behaviour in real life as well as in laboratory or office settings, interviews and self-report measures and various other methods of cognitive assessment (Bellack & Hersen, 1998). We turn to these now.

### Direct observation of behaviour

In formal behavioural observation, the observer divides the sequence of behaviour into various parts that make sense within a learning framework, including such things as the antecedents and consequences of particular behaviours. Behavioural observation is also often linked to intervention (O'Brien & Haynes, 1995).

It is difficult to observe most behaviour as it actually takes place and little control can be exercised over where and when it may occur. For this reason, many therapists contrive somewhat artificial situations in their consulting rooms or in a laboratory so that they can observe how a person or a family acts under certain conditions. For example, Barkley (1981) had a mother and her child spend time together in a laboratory living room, complete with sofas and a television. The mother was given a list of tasks for the child to complete, such as picking up toys or doing maths problems. Observers behind a one-way mirror watched the proceedings and reliably coded the child's reactions to the mother's efforts to control as well as the mother's reactions to the child's compliant or non-compliant responses. These **behavioural assessment** procedures yielded data that could be used to measure the effects of treatment.

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Behavioural assessment often involves direct observation of behaviour, as in this case, where the observer is behind a one-way mirror.



### Self-observation

Cognitive behaviour therapists and researchers often ask people to observe and track their own behaviour and responses, an approach called **self-monitoring**. Self-monitoring is used to collect a wide variety of data, including moods, stressful experiences, coping behaviours and thoughts (Hurlburt, 1979; Stone, Schwartz, Neale et al., 1998).

Another method of self-observation is called **ecological momentary assessment (EMA)**. EMA involves the collection of data in real time as opposed to the more usual methods of having people reflect back over some time period and report on recently experienced thoughts, moods or stressors. With EMA, a person is signalled (via text message or smartphone alert most typically) several times a day and asked to enter responses directly into the device (Stone & Shiffman, 1994).

EMA can be useful in clinical settings, revealing information that traditional assessment procedures might miss. For example, Hurlburt (1997) describes a case of a man with severe panic attacks. In clinical interviews, the man reported that his life was going very well, that he loved his wife and children and that his work was both financially and personally rewarding. No cause of the panic attacks could be discerned. The man was asked to record his thoughts as he went about his daily routine. Surprisingly, about a third of his thoughts were concerned with annoyance with his children (e.g., 'He left the fence gate open again and the dog got out').

Once the high frequency of annoyance thoughts was pointed out to him, he ... accepted that he was in fact often annoyed with his children. However, he believed that anger at his children was sinful and felt unfit as a father for having such thoughts and feelings ... [He] entered into brief therapy that focused on the normality of being annoyed by one's children and on the important distinction between being annoyed and acting out aggressively. Almost immediately, his anxiety attacks disappeared. (1997, p. 944)

Although some research indicates that self-monitoring or EMA can provide accurate measurement of such behaviour, considerable research indicates that behaviour may be altered by the very fact that it is being self-monitored — that is, the self-consciousness required for self-monitoring affects the behaviour (Haynes & Horn, 1982). The phenomenon wherein behaviour changes because it is being observed is called **reactivity**. In general, desirable behaviour, such as engaging in social conversation, often increases in frequency when it is self-monitored (Nelson, Lipinski, & Black, 1976), whereas behaviour a person wishes to reduce, such as cigarette smoking, diminishes (McFall & Hammen, 1971). Therapeutic interventions can take advantage of the reactivity that is a natural by-product of self-monitoring. Smoking, anxiety, depression and health problems have all undergone beneficial changes in self-monitoring studies (Febbraro & Clum, 1998). Beyond reactivity, self-monitoring with portable electronic devices like smartphones has also been included effectively in cognitive-behavioural therapy for different anxiety disorders (Przeworski & Newman, 2006).

### Cognitive-style questionnaires

Cognitive questionnaires tend to be used to help plan targets for treatment as well as to determine whether clinical interventions are helping to change overly negative thought patterns as well as negative and positive emotions. In format, some of these questionnaires are similar to the personality tests we have already described.

One self-report questionnaire that was developed based on Beck's theory (see chapter 1 and the chapter on mood disorders) is the Dysfunctional Attitude Scale (DAS). The DAS contains items such as 'People will probably think less of me if I make a mistake' (Weissman & Beck, 1978). Supporting construct validity, researchers have shown that they can differentiate between people with and without depression on the basis of their scores on this scale and that scores decrease (i.e., improve) after interventions that relieve depression. Furthermore, the DAS relates to other aspects of cognition in ways consistent with Beck's theory (Glass & Arnkoff, 1997).

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Self-monitoring generally leads to increases in desirable behaviours and decreases in undesirable ones.



**TABLE 2.6** Psychological assessment methods

<b>Interviews</b>	Clinical interviews	The clinician learns about the person's problems through conversation. The paradigm of the interviewer shapes the content of the interview.
	Structured interviews	Questions to be asked are spelled out in detail in a booklet. Structured clinical interviews are commonly used to make a diagnosis.
<b>Stress measures</b>		Self-report scales or interviews that assess stressful events and responses to these events.
<b>Psychological tests</b>	Personality tests	Self-report questionnaires, used to assess either a broad range of characteristics, as in the MMPI-2 or a single characteristic, such as dysfunctional attitudes.
	Projective tests	Ambiguous stimuli, such as inkblots (Rorschach test), are presented and responses are thought to be determined by unconscious processes.
	Intelligence tests	Assessments of current mental functioning. Used to predict school performance and identify cognitive strengths and weaknesses.
<b>Direct observation</b>		Used by clinicians to identify problem behaviours as well as antecedents and consequences.
<b>Self-observation</b>		People monitor and keep records of their own behaviour, as in ecological momentary assessment.

Cognitive assessment focuses on the person's perception of a situation, realising that the same event can be perceived differently. For example, house hunting could be regarded as a very negative event or a very positive one, resulting in very different levels of stress.



## 2.4 Neurobiological assessment

**LEARNING OUTCOME 2.4** Describe the goals, strengths and weaknesses of neurobiological approaches to assessment.

Recall from chapter 1 that throughout history people interested in psychopathology have assumed, quite reasonably, that some symptoms are likely to be due to or at least reflected in malfunctions of the brain. We turn now to contemporary work in neurobiological assessment. We'll look at four areas in particular: brain imaging, neurotransmitter assessment, neuropsychological assessment and psychophysiological assessment (see table 2.7 for a summary of these methods).

**TABLE 2.7** Neurobiological assessment methods

Brain imaging	CT and MRI scans reveal the structure of the brain. PET reveals brain function and, to a lesser extent, brain structure. fMRI is used to assess both brain structure and brain function.
Neurotransmitter assessment	Includes postmortem analysis of neurotransmitters and receptors, assays of metabolites of neurotransmitters and PET scans of neurotransmitter receptors.
Neuropsychological assessment	Behavioural tests such as the Halstead–Reitan and Luria–Nebraska assess abilities such as motor speed, memory and spatial ability. Deficits on particular tests help point to an area of possible brain dysfunction.
Psychophysiological assessment	Includes measures of electrical activity in the autonomic nervous system, such as skin conductance or in the central nervous system, such as EEG.

### Brain imaging: 'seeing' the brain

Because many behavioural problems can be brought on by brain dysfunction, neurological tests — such as checking the reflexes, examining the retina for any indication of blood vessel damage and evaluating motor coordination and perception — have been used for many years to identify brain dysfunction. Today, devices have become available that allow clinicians and researchers a much more direct look at both the structure and functioning of the brain.

Computerised axial tomography, the **CT** or **CAT scan**, helps to assess structural brain abnormalities (and is able to image other parts of the body for medical purposes). A moving beam of x-rays passes into a horizontal cross-section of the person's brain, scanning it through 360 degrees; the moving x-ray detector on the other side measures the amount of radioactivity that penetrates, thus detecting subtle differences in tissue density. A computer uses the information to construct a two-dimensional, detailed image of the cross-section, giving it optimal contrasts. Then the machine scans another cross-section of the brain. The resulting images can show the enlargement of ventricles (which can be a sign of brain tissue degeneration) and the locations of tumours and blood clots.

Other devices for seeing the living brain include **magnetic resonance imaging**, also known as **MRI**, which is superior to the CT scan because it produces pictures of higher quality and does not rely on even the small amount of radiation required by a CT scan. In MRI the person is placed inside a large, circular magnet, which causes the hydrogen atoms in the body to move. When the magnetic force is turned off, the atoms return to their original positions and thereby produce an electromagnetic signal. These signals are then read by the computer and translated into pictures of brain tissue. This technique provides an enormous advance. For example, it has allowed physicians to locate delicate brain tumours that would have been considered inoperable without such sophisticated methods of viewing brain structures.

An even greater advance has been a technique called **functional magnetic resonance imaging (fMRI)**, which allows researchers to measure both brain structure and brain function. This technique takes MRI pictures so quickly that metabolic changes can be measured, providing a picture of the brain

at work rather than of its structure alone. fMRI measures blood flow in the brain and this is called the **BOLD** signal, which stands for **blood oxygenation level dependent**. As neurons fire, presumably blood flow increases to that area. Therefore, blood flow in a particular region of the brain is a reasonable proxy for neural activity in that brain region.

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A person entering an fMRI scanner



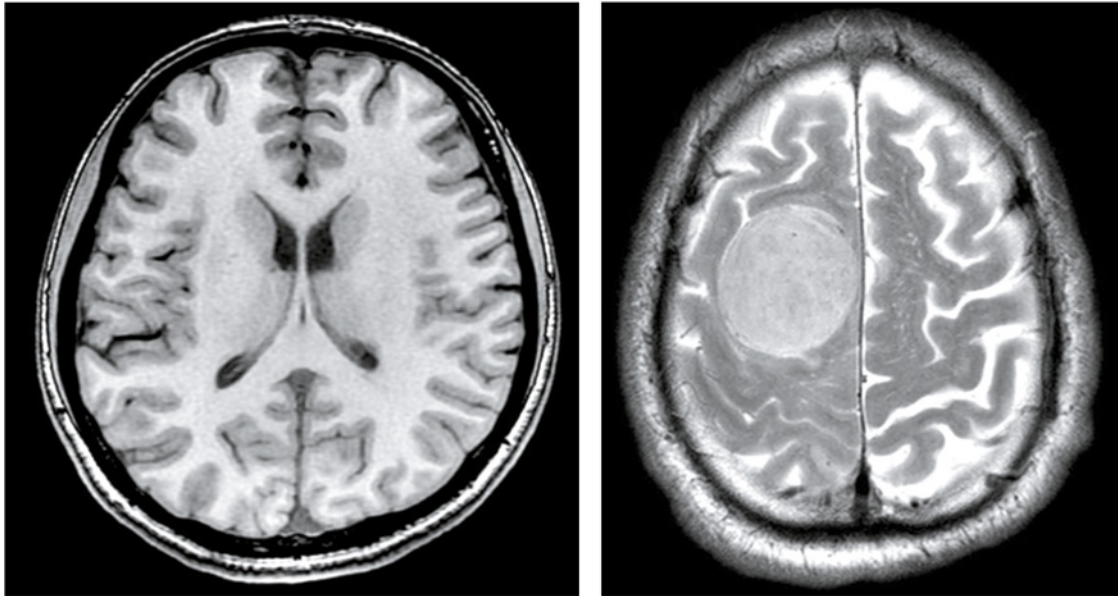
Positron emission tomography, the **PET scan**, a more expensive and invasive procedure, also allows measurement of both brain structure and brain function, although the measurement of brain structure is not as precise as with MRI or fMRI. A substance used by the brain is labelled with a short-lived radioactive isotope and injected into the bloodstream. The radioactive molecules of the substance emit a particle called a positron, which quickly collides with an electron. A pair of high-energy light particles shoot out from the skull in opposite directions and are detected by the scanner. The computer analyses millions of such recordings and converts them into a picture of the functioning brain. The images are in colour; fuzzy spots of lighter and warmer colours are areas in which metabolic rates for the substance are higher. Because this is more invasive than fMRI, it is now used less often as a measure of brain function.

Visual images of the working brain can indicate areas of degeneration, tumours, strokes and trauma from head injuries, as well as disruptions in the neural connections between different brain regions, differences in volume (size) of various brain regions and the distribution of psychoactive drugs in the brain. fMRI and to a lesser extent PET are being used to study neural deficits that are linked to various disorders, such as the failure of the prefrontal cortex of people with schizophrenia to become strongly activated while they attempt to perform a cognitive task. Current neuroimaging studies in psychopathology are attempting to identify not only areas of the brain that may be dysfunctional (e.g., the prefrontal cortex) but also deficits in the ways in which different areas of the brain

communicate and connect with one another. This type of inquiry is often referred to as functional connectivity analysis since it aims to identify how different areas of the brain are connected with one another.

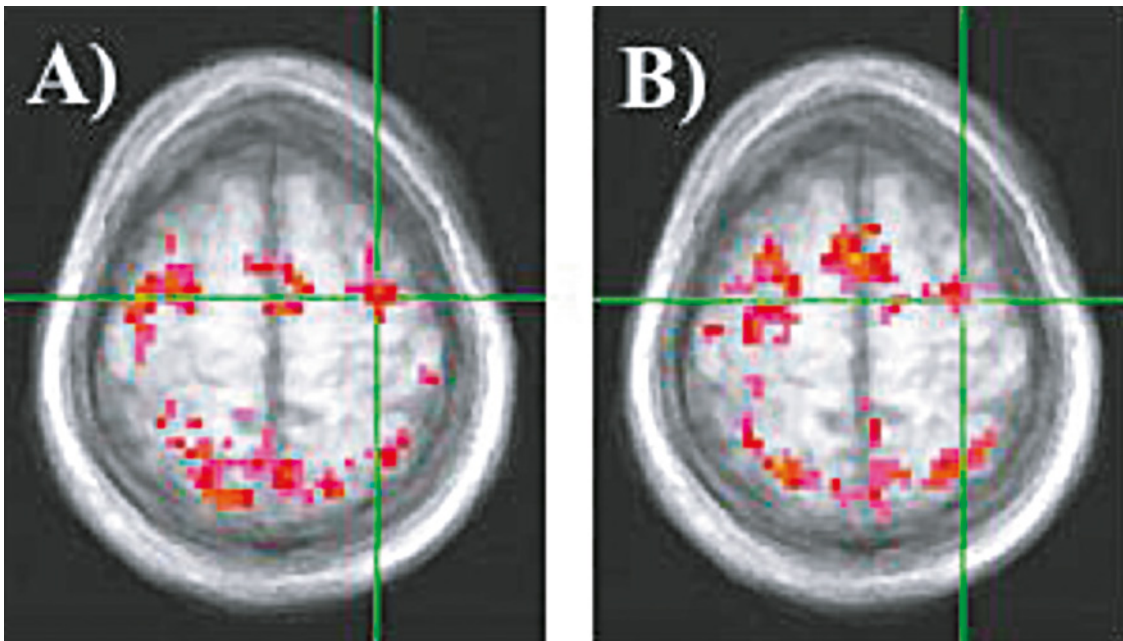
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These two CT scans show a horizontal 'slice' through the brain. The one on the left is normal; the one on the right has a tumour on the left side.



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Functional magnetic resonance images (fMRI). With this method, researchers can measure how brain activity changes while a person is doing different tasks, such as viewing an emotional film, completing a memory test, looking at a visual puzzle or hearing and learning a list of words.



## Neurotransmitter assessment

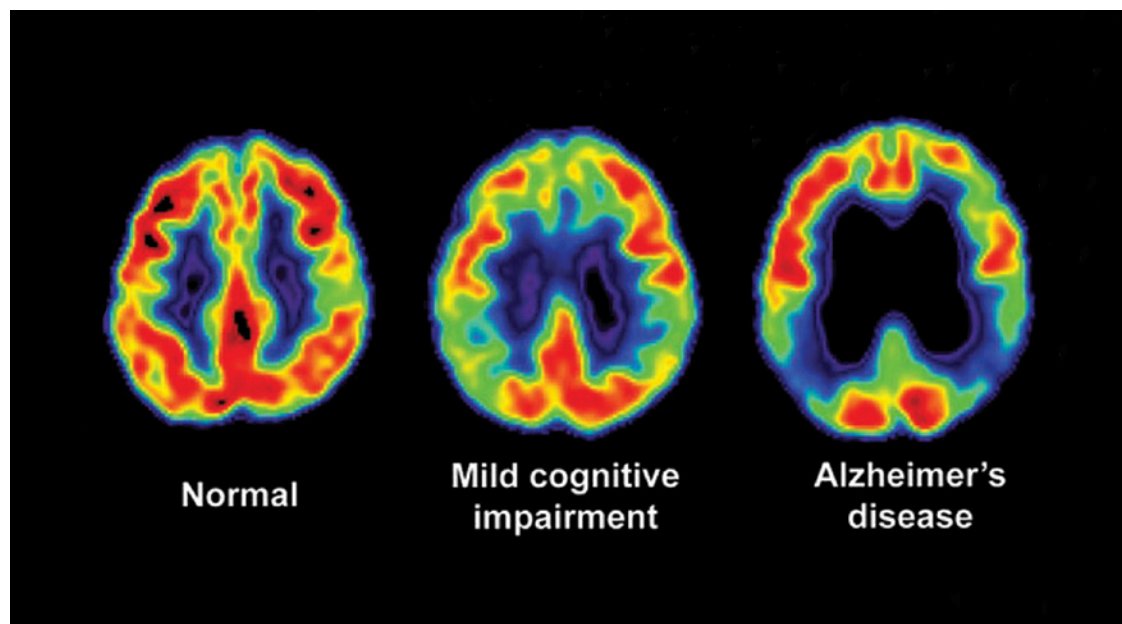
It might seem that assessing the amount of a particular neurotransmitter or the quantity of its receptors in the brain would be straightforward. But as we began to discuss in chapter 1, it is not. Most of the research on neurotransmitters and psychopathology has relied on indirect assessments.

In postmortem studies, the brains of deceased people are removed and the amount of specific neurotransmitters in particular brain areas can then be directly measured. Different brain areas can be infused with substances that bind to receptors and the amount of binding can then be quantified; more binding indicates more receptors.

In studies of participants who are alive, one common method of neurotransmitter assessment involves analysing the metabolites of neurotransmitters that have been broken down by enzymes. A **metabolite**, typically an acid, is produced when a neurotransmitter is deactivated. These by-products of the breakdown of neurotransmitters, such as norepinephrine, dopamine and serotonin, are found in urine, blood serum and cerebrospinal fluid (CSF; the fluid in the spinal column and in the brain's ventricles). For example, a major metabolite of dopamine is homovanillic acid (HVA); that of serotonin is 5-hydroxyindoleacetic acid (5-HIAA). A high level of a particular metabolite presumably indicates a high level of a neurotransmitter and a low level indicates a low level of the neurotransmitter.

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The PET scan on the left shows a normal brain; the one in the middle shows a brain with mild cognitive impairment; the one on the right shows the brain of a person with Alzheimer's disease.



But there is a problem with measuring metabolites from blood or urine: such measures are not direct reflections of levels of neurotransmitters in the brain; metabolites measured in this way could reflect neurotransmitters anywhere in the body. A more specific measure can be taken of metabolites in the CSF fluid drawn from a person's spinal cord. Even with CSF fluid, however, metabolites reflect activity throughout the brain and spinal cord, rather than regions that are directly involved in psychopathology. Studies of 5-HIAA, the main metabolite of serotonin, and depression have been inconsistent, perhaps owing to the fact that these metabolites do not directly reflect serotonin levels of activity.

Another problem with metabolite studies is that they are correlational. Causation cannot be determined from a correlational study. That is, when researchers find that neurotransmitter levels are low

among people with a particular disorder, such as depression, this could be because neurotransmitter levels cause depression, because depression causes neurotransmitter changes or because a third variable causes shifts in both neurotransmitters and depression. For example, dopamine, norepinephrine and serotonin levels change in response to stress. To test whether neurotransmitter levels could cause symptoms, experimental evidence is needed.

To provide more experimental data on whether these neurotransmitter systems actually help cause psychopathology, one strategy is to administer drugs that increase or decrease levels of neurotransmitters. For example, a drug that raises the level of serotonin should alleviate depression; one reducing it should trigger depressive symptoms. This strategy also has its problems, though. One might wonder whether it is ethical to conduct these studies if the goal of an experiment is to produce symptoms. Another issue is that drugs that change levels of one neurotransmitter often tend to influence other neurotransmitter systems. We will see examples of these types of studies throughout this text.

Clinicians and researchers in many disciplines are currently using brain-imaging and neurotransmitter assessment techniques both to discover previously undetectable brain problems and to conduct inquiries into the neurobiological contributions to thought, emotion and behaviour. It is a very lively and exciting area of research and application. Indeed, one might reasonably assume that researchers and clinicians, with the help of such procedures and technological devices as fMRI, could observe the brain and its functions more or less directly and thus assess all brain abnormalities. Results to date, however, are not strong enough for these methods to be used in diagnosing psychopathology. Moreover, many brain abnormalities involve alterations in structure so subtle or slight in extent that they have thus far eluded direct examination. Furthermore, the problems in some disorders are so widespread that finding the contributing brain dysfunction is a daunting task. Take, for example, schizophrenia, which affects thinking, feeling and behaviour. Where in the brain might there be dysfunction? Looking for areas that influence thinking, feeling and behaviour requires looking at just about the entire brain.

## Neuropsychological assessment

It is important at this point to note a distinction between neurologists and neuropsychologists, even though both specialists are concerned with the study of the central nervous system. A **neurologist** is a physician who specialises in diseases or problems that affect the nervous system, such as stroke, muscular dystrophy, cerebral palsy or Alzheimer's disease. A **neuropsychologist** is a psychologist who studies how dysfunctions of the brain affect the way we think, feel and behave. Both kinds of specialists contribute much to each other as they work in different ways, often collaboratively, to learn how the nervous system functions and how to ameliorate problems caused by disease or injury to the brain.

**Neuropsychological tests** are often used in conjunction with the brain-imaging techniques just described, both to detect brain dysfunction and to help pinpoint specific areas of behaviour that are impacted by problems in the brain. Neuropsychological tests are based on the idea that different psychological functions (e.g., motor speed, memory, language) rely on different areas of the brain. Thus, for example, neuropsychological testing might help identify the extent of brain damage suffered during a stroke and it can provide clues about where in the brain the damage may exist that can then be confirmed with more expensive brain-imaging techniques. There are numerous neuropsychological tests used in psychopathology assessment. Here, we highlight two widely used batteries of tests.

One neuropsychological test is Reitan's modification of a battery, or group, of tests previously developed by Halstead, called the Halstead–Reitan neuropsychological test battery. The following are three of the Halstead–Reitan tests.

1. *Tactile Performance Test — Time*. While blindfolded, the person tries to fit variously shaped blocks into spaces of a form board, first using the preferred hand, then the other, and finally both.
2. *Tactile Performance Test — Memory*. After completing the timed test, the person is asked to draw the form board from memory, showing the blocks in their proper location. Both this and the timed test are sensitive to damage in the right parietal lobe.

3. *Speech Sounds Perception Test*. Participants listen to a series of nonsense words, each comprising two consonants with a long-*e* sound in the middle. They then select the ‘word’ they heard from a set of alternatives. This test measures left-hemisphere function, especially temporal and parietal areas.

Extensive research has demonstrated that the battery is valid for detecting behaviour changes linked to brain dysfunction resulting from a variety of conditions, such as tumours, stroke and head injury (Horton, 2008).

The Luria–Nebraska battery (Golden, Hammeke, & Purisch, 1978), based on the work of the Russian psychologist Aleksandr Luria (1902–1977), is also widely used (Moses & Purisch, 1997). The battery includes 269 items divided into 11 sections designed to determine basic and complex motor skills, rhythm and pitch abilities, tactile and kinaesthetic skills, verbal and spatial skills, receptive speech ability, expressive speech ability, writing, reading, arithmetic skills, memory and intellectual processes. The pattern of scores on these sections, as well as on the 32 items found to be the most discriminating and indicative of overall impairment, helps reveal potential damage to the frontal, temporal, sensorimotor or parietal-occipital area of the right or left hemisphere.

The Luria–Nebraska battery can be administered in  $2\frac{1}{2}$  hours and can be scored in a highly reliable manner (e.g., Kashden & Franzen, 1996). Criterion validity has been established by findings that test scores can correctly distinguish 86 percent of people with and without neurological disease (Moses, Schefft, Wong, & Berg, 1992). A particular advantage of the Luria–Nebraska tests is that one can control for educational level so that a less educated person will not receive a lower score solely because of limited educational experience (Brickman, McManus, Grapentine, & Alessi, 1984). Finally, a version for children aged 8 to 12 (Golden, 1981a, 1981b) has been found useful in helping to pinpoint brain damage and in evaluating the educational strengths and weaknesses of children (Sweet, Carr, Rossini, & Kasper, 1986).

## Psychophysiological assessment

The discipline of **psychophysiology** is concerned with the bodily changes that are associated with psychological events. Experimenters have used measures such as heart rate, tension in the muscles, blood flow in various parts of the body and electrical activity in the brain (so-called brain waves) to study physiological changes when people are afraid, depressed, asleep, imagining, solving problems and so on. Like the brain-imaging methods we have already discussed, the assessments we describe here are not sensitive enough to be used for diagnosis. They can, however, provide important information about a person’s reactivity and can also be used to compare individuals. For example, in using exposure to treat a person with an anxiety disorder, it would be useful to know the extent to which the person shows physiological reactivity when exposed to the stimuli that create anxiety. People who show more physiological reactivity may be experiencing more fear, which predicts more benefit from the therapy (Foa, Riggs, Marsie, & Yarczower, 1995).

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Measures of neurotransmitter metabolites in blood or urine levels do not provide a very accurate index of neurotransmitter levels in the brain.



The activities of the autonomic nervous system (also discussed in chapter 1) are often assessed to understand aspects of emotion. One such measure is heart rate. Each heartbeat generates electrical changes, which can be recorded by electrodes placed on the chest that convey signals to an electrocardiograph or a polygraph. The signal is graphically depicted in an **electrocardiogram (EKG)**, which may be seen as waves on a computer screen.

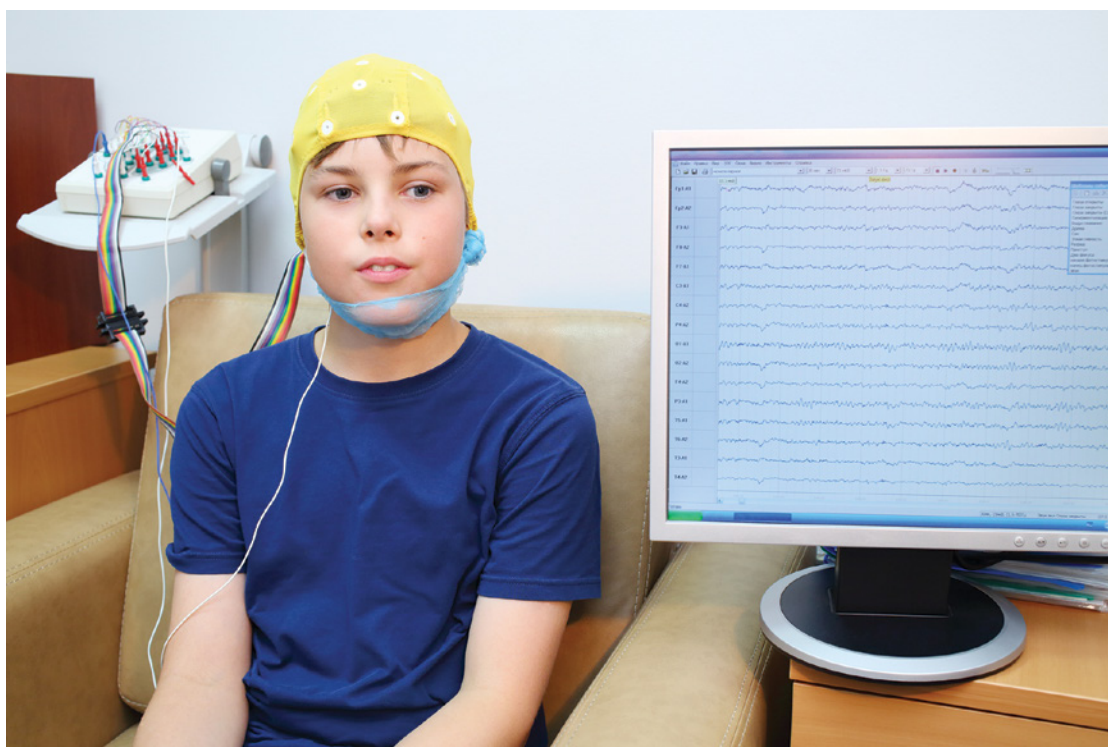
A second measure of autonomic nervous system activity is **electrodermal responding** or skin conductance. Anxiety, fear, anger and other emotions increase activity in the sympathetic nervous system, which then boosts sweat-gland activity. Increased sweat-gland activity increases the electrical conductance of the skin. Conductance is typically measured by determining the current that flows through the skin as a small voltage is passed between two electrodes on the hand. When the sweat glands are activated, this current shows a pronounced increase. Since the sweat glands are activated by the sympathetic nervous system, increased sweat-gland activity indicates sympathetic autonomic excitation and is often taken as a measure of emotional arousal. These measures are widely used in research in psychopathology.

Brain activity can be monitored by **electroencephalography**. Electrodes placed on the scalp record electrical activity in the underlying brain area. Abnormal patterns of electrical activity can indicate seizure activity in the brain or help in locating brain lesions or tumours. EEG indices are also used to measure attention and alertness.

As with the brain-imaging techniques reviewed earlier, a more complete picture of a human being is obtained when physiological functioning is assessed while the person is engaging in some form of behaviour or cognitive activity. If researchers and clinicians are interested in psychophysiological responding in people with a specific phobia of spiders, for example, they would likely study the people while presenting stimuli, such as pictures of spiders or even actual spiders that would elicit the anxiety and fear responses.

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Electroencephalography measures brain activity by recording electrical activity in the underlying brain area.

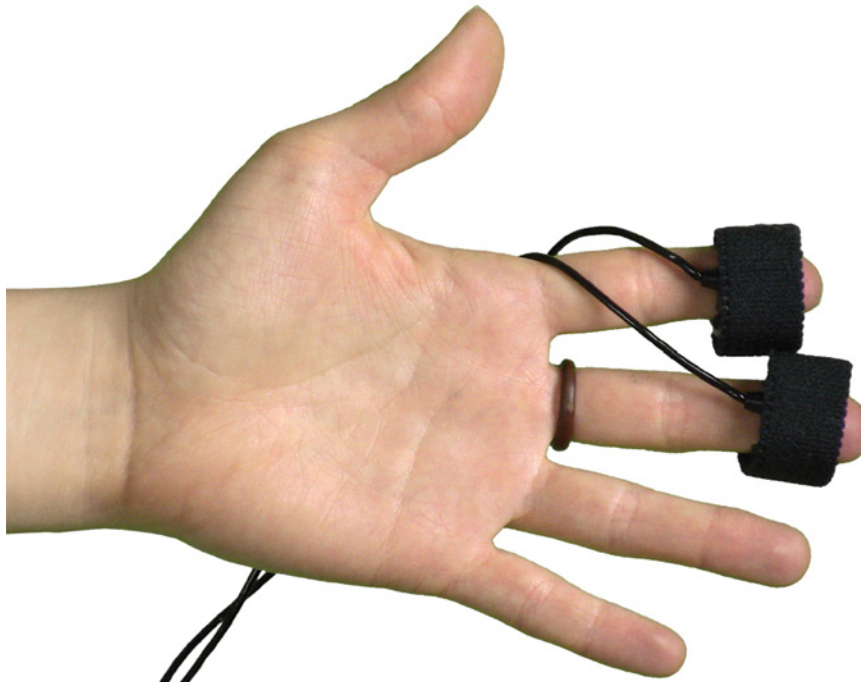


## A cautionary note about neurobiological assessment

A cautionary note regarding neurobiological assessment methods is in order here. Inasmuch as psychophysiology and brain imaging employ highly sophisticated electronic machinery and many psychologists aspire to be as scientific as possible, researchers and clinicians sometimes believe uncritically in these apparently objective assessment devices without appreciating their real limitations and complications. Many of the measurements do not differentiate clearly among emotional states. Skin conductance, for example, increases not only with anxiety but also with other emotions — among them, happiness. In addition, being in a scanner is often a threatening experience. Thus, the investigator interested in measuring brain changes associated with emotion using fMRI must also take the scanning environment into account. It is also important to keep in mind that brain-imaging techniques do not allow us to manipulate brain activity and then measure a change in behaviour (Feldman Barrett, 2003). In a typical study, we show people a list of emotionally evocative words and then measure blood flow in the brain. Does a person who fails to show the same level of activation in emotion regions during this task have a brain-based emotion deficit? Not necessarily. The person might not have paid attention, might not have understood the words or might be focused on the loud clanging noises that the fMRI machine is making. It is important to be extremely careful in considering alternative explanations for the effects found in these studies.

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In psychophysiological assessment, physical changes in the body are measured. Skin conductance can be measured with sensors on two fingers.



Neither is there a one-to-one relationship between a score on a given neuropsychological test or a finding on an fMRI scan on the one hand and psychological dysfunction on the other. The reasons for these sometimes loose relationships have to do with such factors as how the person has, over time, developed different coping strategies. And the success of coping, in turn, has to do with the social environment in which the person has lived, for example, how understanding parents and friends have been or how well the school system has provided for the special educational needs of the person. Furthermore, the brain itself will change in response to these psychological and socioenvironmental factors over time. Therefore, in addition to the imperfect nature of the neurobiological assessment instruments themselves

and our incomplete understanding of how the brain actually functions, clinicians and researchers must consider social and environmental factors that operate over time to contribute to the clinical picture. In other words, a complete assessment must include multiple methods (clinical interviews, psychological and neurobiological methods).

A final caution is reflected in the simple yet often unappreciated fact that in attempting to understand the consequences of any brain dysfunction, one must understand the pre-existing abilities that the person had prior to diagnosis with a psychological disorder. This straightforward truth brings to mind the story of the man who, recovering from an accident that has broken all the fingers in both hands, earnestly asks the surgeon whether he will be able to play the piano when his wounds heal. 'Yes, I'm sure you will,' says the doctor reassuringly. 'That's wonderful,' exclaims the man, 'I've always wanted to be able to play the piano'.

## 2.5 Cultural and ethnic diversity and assessment

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**LEARNING OUTCOME 2.5** Discuss the ways in which culture and ethnicity impact diagnosis and assessment.

Studies of the influences of culture and ethnicity on psychopathology and its assessment have proliferated in recent years. As you read about some of this research, it is critical to keep in mind that there are typically more differences within cultural, ethnic and racial groups than there are between them. Remembering this important point can help you avoid the dangers of stereotyping members of a culture or ethnic group.

We should also note that the reliability and validity of various forms of psychological assessment have been questioned on the grounds that their content and scoring procedures reflect the culture of white European Americans and so may not accurately assess people from other cultures. In this section we discuss problems of cultural bias and what can be done about them.

### Cultural bias in assessment

The issue of cultural bias in assessment refers to the notion that a measure developed for one culture or ethnic group may not be equally reliable and valid with a different cultural or ethnic group. Some tests that were developed in the United States have been translated into different languages and used in different cultures successfully. For example, the WAIS-IV includes a revised normative sample of 2200 adults stratified by age, gender, education level and ethnicity. The WAIS has also been translated and adapted for use in 20 different countries. Additionally, the MMPI-2 has been translated into more than two dozen languages (Tsai, Butcher, Vitousek, & Munoz, 2001). In 2008 the Australian and New Zealand Language Adapted Edition was released. Despite this, a cognitive assessment tool standardised for use with Indigenous Australians is needed (Dingwall, Pinkerton, & Lindeman, 2013).

Simply translating words into a different language, however, does not ensure that the meaning of those words will be the same across different cultures. Several steps in the translation process, including working with multiple translators, back-translating and testing with multiple native speakers, can help to ensure that the test is similar in different languages. This approach has been successful in achieving equivalence across different cultures and ethnic groups for some instruments, such as the MMPI-2 (Arbisi, Ben-Porath, & McNulty, 2002). Even with the MMPI-2, however, there are cultural differences that are not likely attributable to differences in psychopathology. For example, among Asian Americans who are not heavily assimilated into American culture, scores on most MMPI-2 scales are higher than those of Caucasians (Tsai & Pike, 2000). This is unlikely to reflect truly higher emotional disturbance among Asians.

Despite these efforts, the field has some way to go in reducing cultural and ethnic bias in clinical assessment. These cultural assumptions or biases may cause clinicians to over- or underestimate psychological problems in members of other cultures (Lopez, 1989, 1996). Indigenous Australians perform, on average, one standard deviation lower than non-Indigenous Australians on IQ tests (Leigh & Gong, 2009). This does not likely reflect an actual difference in intellectual functioning but instead a form of

ethnic bias in the nature of these tests and on the part of clinicians (Dingwall et al., 2013; Arnold et al., 2004; Trierweiler et al., 2000). Yet take the example of an Asian Australian man who is very emotionally withdrawn. Should the clinician consider the notion that Asian cultures take a more positive view of lower emotional expressiveness in men than do European cultures? A clinician who quickly attributes the behaviour to a cultural difference may overlook an emotional problem that he or she would be likely to diagnose if the person were a white male. Indeed, recent evidence shows that clinicians confuse the absence of excitement in the facial expressions of Asian people as a sign of depression. Even though Asians are less likely to show such expressions than European Americans, this has nothing to do with whether or not they have depression (Tsai, 2014).

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Assessment must take the person's cultural background into account. Belief in spirit possession in some cultures should not always be taken to mean that the believer is psychotic.



How do such biases come about? Cultural factors may affect assessment in various ways. Language differences, differing religious and spiritual beliefs, the alienation or presumed timidity of members of ethnic groups when being assessed by clinicians of different cultural background — all these factors can play a role. For example, clinicians who encounter people claiming to be surrounded by spirits might view this belief as a sign of schizophrenia. Yet in Puerto Rican cultures, such a belief is common; therefore, believing that one is surrounded by spirits should probably not be taken as a sign of schizophrenia in a Puerto Rican person (Rogler & Hollingshead, 1985).

Cultural and ethnic differences in psychopathology must be examined more closely. Unfortunately, the cultural and ethnic biases that can creep into clinical assessment do not necessarily yield efforts to compensate for them. There is no simple answer. The DSM-5's emphasis on cultural factors in the discussion of every category of disorder, along with its new cultural formulation interview, may well sensitise clinicians and researchers to the issue. When practitioners were surveyed, they overwhelmingly reported taking culture into account in their clinical work (Lopez, 1994), so it appears that the problem, if not the solution, is clearly in focus.

## Strategies for avoiding cultural bias in assessment

Clinicians can — and do — use various methods to minimise the negative effects of cultural biases when conducting assessments. Perhaps the place to begin is with graduate training programs. Lopez (2002) has noted three important issues that should be taught to graduate students in clinical psychology programs. First, students must learn about basic issues in assessment, such as reliability and validity. Second, students must become informed about the specific ways in which culture or ethnicity may impact assessment rather than rely on more global stereotypes about a particular cultural or ethnic group. Third, students must consider that culture or ethnicity may not impact assessment in every individual case. Indeed, the Australian Psychology Accreditation Council requires all psychology graduates in Australia to have received training in cultural competence, including with Aboriginal and Torres Strait Islander cultures.

Finally, when the examiner and client have different ethnic backgrounds, the examiner may need to make an extra effort to establish a rapport that will result in the person's best performance. To assist with this, the Australian Psychological Society has published *Guidelines for the Provision of Psychological Services for, and the Conduct of Psychological Research with, Aboriginal and Torres Strait Islander People of Australia* (APS, 2003).

As Lopez (1994) points out, however, 'the distance between cultural responsiveness and cultural stereotyping can be short' (p. 123). To minimise such problems, clinicians are encouraged to be particularly tentative about drawing conclusions regarding people from different cultural and ethnic backgrounds. Rather, they are advised to make hypotheses about the influence of culture on a particular person, entertain alternative hypotheses and then test those hypotheses. Training in cultural awareness is truly important, as a clinician's biases can influence diagnosis. One way to combat these biases is to use structured diagnostic interviews, like the SCID described earlier. When clinicians use structured interviews, they are less likely to over-diagnose people from different ethnic groups (Garb, 2005).

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Cultural differences can lead to different results on an aptitude or IQ test. For example, in New Zealand, Maori students have lower educational attainment levels than their non-Indigenous counterparts.



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## SUMMARY

### 2.1 Distinguish the different types of reliability and validity.

Because diagnosis can provide the first step in thinking about the causes of symptoms, it is often the first step in planning treatment. Because psychopathology is diagnosed on the basis of symptoms, clinical interviews are used to make diagnoses.

With all assessments, the reliability (the consistency of measurement) and validity (whether an assessment measures what it is designed to measure) should be evaluated. Reliability can be estimated by examining how well raters agree, how consistent test scores are over time, how alternate forms of a test compare or how well items correlate with each other. There are many different forms of validity, including content, criterion and construct validity.

Diagnostic systems for psychological disorders have changed a great deal in the past 100 years. Drawing on research evidence, the order of chapters in the DSM-5 is reorganised to reflect current knowledge of aetiology. Some disorders are added, some are removed and others are combined. DSM-5 continues to emphasise culturally sensitive case formulations.

### 2.2 Identify the basic features, historical changes, strengths and weaknesses of the DSM.

Despite improvements in the DSM-5, a number of problems remain. Some argue that there are too many diagnoses. Others challenge the use of a categorical rather than a dimensional approach. Reliability is higher than it was in earlier editions of the DSM, but clinicians still disagree regarding some diagnoses and the reliability achieved in practice may not be as high as the reliability achieved in research studies. Finally, the field as a whole faces a huge challenge; researchers are focused on validating this diagnostic system by trying to identify the causal patterns, symptom patterns and treatment that can be predicted by a given diagnosis. In sum, although the DSM is continually improving, it is far from perfect. Regardless of which diagnostic system is used, there are certain problems inherent in diagnosing people with psychological disorders. It is important to be aware of the tendency to ignore a person's strengths when focusing on diagnoses. The American Psychological Association recommends using phrases such as *person with schizophrenia* rather than *schizophrenic* as one way to acknowledge that a person is much more than his or her diagnosis. Although many worry that applying labels may increase stigma, diagnoses can sometimes relieve stigma by providing a way of understanding symptoms.

### 2.3 Describe the goals, strengths and weaknesses of psychological approaches to assessment.

The psychological assessments we have described are summarised in table 2.6. A comprehensive psychological assessment draws on many different methods and tests. Interviews can be structured, with the questions predetermined and followed in a certain order, or unstructured, to follow more closely what the person tells the interviewer. Structured interviews are more reliable. Rapport is important to establish regardless of the type of interview.

Stress is best assessed via a semistructured interview that captures the importance of any given life event in the context of a person's life circumstances, as in the LEDS. Self-report checklists are also used to assess stress, but they have poorer reliability and validity than the LEDS.

The MMPI-2 is a standardised and objective personality inventory. The test has good reliability and validity and is widely used. Projective personality tests, like the Rorschach or TAT, are not as widely used today, likely due to their poor validity. Reliability can be achieved using scoring systems such as Exner's. Intelligence tests have been used for a number of years and are quite reliable. Like any test, there are limits to what an IQ test can tell a clinician or researcher.

Direct observation of behaviour can be very useful in assessment, though it can take more time than a self-report inventory. Other behavioural and cognitive assessment methods include ecological momentary assessment (EMA) and questionnaires.

## 2.4 Describe the goals, strengths and weaknesses of neurobiological approaches to assessment.

Advances in technology have allowed clinicians and researchers to 'see' the living brain. Different imaging techniques, such as CT, MRI and fMRI, have the potential to show areas of the brain that might not be working optimally. Direct assessment of neurotransmitters is not done often. Rather, examinations of the metabolites of neurotransmitters provide a rough way to estimate how neurotransmitters are functioning. Another approach is to administer drugs that increase or decrease the levels of a neurotransmitter. Postmortem exams also allow for measurements of neurotransmitters, particularly receptors. Neuropsychological tests are tests that have been developed to show how changes in behaviour may reflect damage or disturbance in particular areas of the brain. Psychophysiological assessment methods can show how behaviours and cognitions are linked to changes in nervous system activity, such as heart rate, skin conductance or brain electrical activity. These methods have as many as or more limitations than other assessment measures, and the key concepts of reliability and validity are just as relevant with neurobiological assessment as with other forms of assessment.

## 2.5 Discuss the ways in which culture and ethnicity impact diagnosis and assessment.

Cultural and ethnic factors play a role in clinical assessment. Assessment techniques developed on the basis of research with Caucasian populations may be inaccurate when used with people of differing ethnic or cultural backgrounds. Clinicians can have biases when evaluating people from different ethnic and cultural groups, which can lead to minimising or exaggerating a person's psychopathology. Clinicians can use various methods to guard against the negative effects of cultural biases in assessment. In gathering diagnosis and assessment information, clinicians and researchers must be concerned with both reliability and validity, and how cultural differences can impact these.

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## KEY TERMS

**alternate-form reliability** the relationship between scores achieved by people when they complete two versions of a test that are judged to be equivalent

**Australian Psychological Society (APS)** the largest professional association for psychologists in Australia

**Australian Psychological Society's Code of Ethics** a code of conduct for psychologists in Australia that is published by the Australian Psychological Society

**behavioural assessment** a sampling of ongoing cognitions, feelings and overt behaviour in their situational context

**BOLD (blood oxygenation level dependent)** the signal detected by functional MRI studies of the brain; measures blood flow and thus neural activity in particular regions

**case conceptualisation** a process of integrating a patient's assessment information with theory and research

**categorical classification** an approach to assessment in which a person is or is not a member of a discrete grouping

**clinical interview** general term for conversation between a clinician and a patient that is aimed at determining diagnosis, history, causes of problems and possible treatment options

**comorbidity** the co-occurrence of two disorders, as when a person has depression and social phobia

**concurrent validity** the extent to which previously undiscovered features are found among patients with the same diagnosis

**construct validity** the extent to which scores or ratings on an assessment instrument relate to other variables or behaviours according to some theory or hypothesis

**content validity** the extent to which a measure adequately samples the domain of interest

**criterion validity** the extent to which a measure is associated in an expected way with some other measure

**CT or CAT scan** refers to computerised axial tomography, a method of diagnosis in which x-rays are taken from different angles and then analysed by computer to produce a representation of the part of the body in cross-section

**diagnosis** the determination that the set of symptoms or problems of a patient indicates a particular disorder

***Diagnostic and Statistical Manual of Mental Disorders (DSM)*** the manual of clinical syndromes published by the American Psychiatric Association and used for descriptive diagnosis. DSM-5 was published in 2013, with major changes to the classification system and diagnostic categories

**dimensional diagnostic system** an approach to classifying mental disorders that involves considering symptoms, behaviours and characteristics on a continuum, rather than assigning them to a category

**ecological momentary assessment (EMA)** form of self-observation involving collection of data in real time (e.g., diaries) regarding thoughts, moods and stressors

**electrocardiogram (EKG)** a recording of the electrical activity of the heart, made with an electrocardiograph

**electrodermal responding** a recording of the minute electrical activity of the sweat glands on the skin, allowing inference of an emotional state

**electroencephalography** a graphic recording of electrical activity of the brain, usually of the cerebral cortex, but sometimes of lower areas

**functional magnetic resonance imaging (fMRI)** modification of magnetic resonance imaging (MRI) that allows researchers to take pictures of the brain so quickly that metabolic changes can be measured, resulting in a picture of the brain at work rather than of its structure alone

**Health Research Council (HRC)** a government organisation in New Zealand that is responsible for managing the government's investment in health research and overseeing the work of research committees

**Human Research Ethics Committees (HRECs)** committees that oversee research involving human participants in Australia

**intelligence test** a standardised means of assessing a person's current mental ability, for example, the Stanford–Binet test or the Wechsler Adult Intelligence Scale

**internal consistency reliability** the degree to which different items of an assessment are related to one another

**interrater reliability** the relationship between the judgements that at least two raters make independently about a phenomenon

**magnetic resonance imaging (MRI)** a technique for measuring the structure (or, in the case of functional magnetic resonance imaging, the activity) of the living brain. The person is placed inside a large circular magnet that causes hydrogen atoms to move; the return of the atoms to their original positions when the current to the magnet is turned off is translated by a computer into pictures of brain tissue

**metabolite** a chemical breakdown product of an endogenous molecule, such as a neurotransmitter, or of an exogenous drug; used to gauge current or recent level of its precursor

**Minnesota Multiphasic Personality Inventory (MMPI)** a lengthy personality inventory that identifies individuals with states such as anxiety, depression, masculinity–femininity and paranoia, through their true–false replies to groups of statements

**neurologist** physician who specialises in medical diseases that affect the nervous system, such as muscular dystrophy, cerebral palsy or Alzheimer's disease

**neuropsychological tests** psychological tests, such as the Luria–Nebraska, which can detect impairment in different parts of the brain

**neuropsychologist** a psychologist who studies how brain dysfunction affects cognition, emotion and behaviour

**New Zealand Psychological Society (NZPS)** the largest professional association for psychologists in New Zealand

**personality inventory** a self-report questionnaire comprising statements assessing habitual behavioural and affective tendencies

**PET scan** computer-generated picture of the living brain, created by analysis of emissions from radioactive isotopes injected into the bloodstream

**predictive validity** the extent to which predictions can be made about the future behaviour of patients with the same diagnosis

**projective hypothesis** the notion that standard but highly unstructured stimuli, as found in the Rorschach assessment's series of inkblots, are necessary to bypass defences in order to reveal unconscious motives and conflicts

**projective test** a psychological assessment device, such as the Rorschach series of inkblots, employing a set of standard but vague stimuli, on the assumption that unstructured material will allow unconscious motivations and fears to be uncovered

**psychological tests** standardised procedures designed to measure performance on a particular task or to assess personality

**psychophysiology** the discipline concerned with the bodily changes that accompany psychological events

**reactivity** the phenomenon wherein behaviour changes because it is being observed

**reliability** the extent to which a test, measurement or classification system produces the same scientific observation each time it is applied

**Research Domain Criteria (RDoC)** a long-term project by the National Institute of Mental Health to develop new ways of classifying psychological disorders based on dimensions of observable behaviour and neurobiological measures

**Rorschach Inkblot Test** a projective test in which the examinee is instructed to interpret a series of 10 inkblots reproduced on cards

**self-monitoring** in behavioural assessment, a procedure whereby the individual observes and reports certain aspects of his or her own behaviour, thoughts or emotions

**standardisation** the process of constructing a normed assessment procedure that meets the various psychometric criteria for reliability and validity

**stress** state of an organism subjected to a stressor; can take the form of increased autonomic activity and in the long term can cause breakdown of an organ or development of a psychological disorder

**structured interview** an interview in which the questions are set out in a prescribed fashion for the interviewer; assists professionals in making diagnostic decisions based on standardised criteria

**test-retest reliability** the relationship between the scores that a person achieves when he or she takes the same test twice

**Thematic Apperception Test (TAT)** a projective test consisting of black-and-white pictures, each depicting a potentially emotion-laden situation, about each of which the examinee is instructed to make up a story

**validity** in research, includes internal, the extent to which results can be confidently attributed to the manipulation of the independent variable, and external, the extent to which results can be generalised to other populations and settings. Validity as applied to psychiatric diagnoses includes concurrent, the extent to which previously undiscovered features are found among patients with the same diagnosis, and predictive, the extent to which predictions can be made about the future behaviour of patients with the same diagnosis. Validity as applied to psychological and psychiatric measures includes content validity, the extent to which a measure adequately samples the domain of interest, and criterion, the extent to which a measure is associated in an expected way with some other measure (the criterion)

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## WEBSITES

1. The American Psychiatric Association has a website devoted to the DSM-5. On this website there are free assessment measures, fact sheets, webinars and updates on the DSM-5. ([www.dsm5.org](http://www.dsm5.org))
2. The Australian Psychological Society website includes information about the practice of psychologists. On this website you will find a copy of the Code of Ethics and practice guidelines. ([www.psychology.org.au](http://www.psychology.org.au))
3. The New Zealand Psychological Society website includes information about the practice of psychologists in New Zealand. ([www.psychology.org.nz](http://www.psychology.org.nz))

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## REFERENCES

- Arbisi, P. A., Ben-Porath, Y. S., & McNulty, J. (2002). A comparison of MMPI-2 validity in African American and Caucasian psychiatric patients. *Psychological Assessment, 14*, 3–15.
- Arnold, L. M., Keck, P. E., Jr., Collins J., Wilson, R., Fleck, D. E., Corey, K. B., Amicone, J., Adebimpe, V. R., & Strakowski, S. M. (2004). Ethnicity and first-rank symptoms in patients with psychosis. *Schizophrenia Research, 67*, 207–212.
- Australian Institute of Health and Welfare. (2010). *Mental health services in Australia 2007–08*. Mental health series no. 12. Cat. no. HSE 88. Canberra: AIHW.
- Australian Psychological Society (APS). (2003). *Guidelines for the provision of psychological services for and the conduct of psychological research with, Aboriginal and Torres Strait Islander people of Australia*. Melbourne: Australian Psychological Society Limited.
- Australian Psychological Society (APS). (2007). *Code of ethics*. Melbourne: Australian Psychological Society Limited.
- Baer, R. A., & Sekirnjak, G. (1997). Detection of underreporting on the MMPI-II in a clinical population. Effects of information about validity scales. *Journal of Personality Assessment, 69*, 555–567.
- Bagby, M. R., Nicholson, R. A., Bacchionchi, J. R., et al. (2002). The predictive capacity of the MMPI-2 and PAI validity scales and indexes to detect coached and uncoached feigning. *Journal of Personality Assessment, 78*, 69–86.
- Barkley, R. A. (1981). *Hyperactive children: A handbook for diagnosis and treatment*. New York: Guilford Press.
- Bellack, A. S., & Hersen, M. (1998). *Behavioral assessment: A practical handbook* (4th ed.). Boston: Allyn & Bacon.
- Blanchard, J. J., & Brown, S. B. (1998). Structured diagnostic interviews. In C. R. Reynolds (Ed.), *Comprehensive clinical psychology, Volume 3, assessment* (pp. 97–130). New York: Elsevier.
- Brickman, A. S., McManus, M., Grapentine, W. L., & Alessi, N. (1984). Neuropsychological assessment of seriously delinquent adolescents. *Journal of the American Academy of Child Psychiatry, 23*, 453–457.
- Brown, G. W., & Harris, T. O. (1978). *The Bedford College life events and difficulty schedule: directory of contextual threat of events*. London: Bedford College University of London.
- Brown, G. W., & Harris, T. O. (1989). Depression. In T. O. Harris & G. W. Brown (Eds.), *Life events and illness* (pp. 49–93). New York: Guilford Press.
- Brugha, T. S., & Cragg, D. (1990). The list of threatening experiences: The reliability and validity of a brief life events questionnaire. *Acta Psychiatrica Scandinavica, 82*, 77–81.
- Butcher, J. N., Dahlstrom, W. G., Graham, J. R., Tellegen, A., & Kraemer, B. (1989). *Minnesota Multiphasic Personality Inventory-2: Manual for administration and scoring*. Minneapolis: University of Minnesota Press.
- Calvin, C. M., Deary, I. J., Fenton, C., Roberts, B. A., Der, G., Leckenby, N., & Batty, G. D. (2010). Intelligence in youth and all-cause-mortality: systematic review with meta-analysis. *International Journal of Epidemiology, 40*, 626–644.
- Canivez, G. L., & Watkins, M. W. (1998). Long-term stability of the Wechsler Intelligence Scale for Children (3rd ed.). *Psychological Assessment, 10*, 285–291.
- Carver, C. S., Johnson, S. L., & Joormann, J. (2008). Serotonergic function, two-mode models of self-regulation, and vulnerability to depression: What depression has in common with impulsive aggression. *Psychological Bulletin, 134*, 912–943.
- Caspi, A., Houts, R. M., Belsky, D. W., et al. (2014). The p factor: One general psychopathology factor in the structure of psychiatric disorders? *Clinical Psychological Science, 2*(2), 119–137.
- Cohen, S., Frank, E., Doyle, W. J., Rabin, B. S., et al. (1998). Types of stressors that increase susceptibility to the common cold in healthy adults. *Health Psychology, 17*, 214–223.
- Cohen, S., Kamarck, T., & Mermelstein, R. (1983). A global measure of perceived stress. *Journal of Health and Social Behavior, 24*, 385–396.
- Cronbach, L. J., & Meehl, P. E. (1955). Construct validity in psychological tests. *Psychological Bulletin, 52*, 281–302.
- Deary, I. J., & Johnson, W. (2010). Intelligence and education: Causal perceptions drive analytic processes and therefore conclusions. *International Journal of Epidemiology, 39*, 1362–1369.

- Dingwall, K. M., Pinkerton, J., & Lindeman, M. A. (2013). People like numbers: a descriptive study of cognitive assessment methods in clinical practice for Aboriginal Australians in the Northern Territory. *BMC Psychiatry*, 13, 13–42. doi:10.1186/1471-244X-13-42
- Dohrenwend, B. P. (2006). Inventorying stressful life events as risk factors for psychopathology: Toward resolution of the problem of intracategory variability. *Psychological Bulletin*, 132, 477–495.
- Dohrenwend, B. S., Krasnoff, L., Askenasy, A. R., & Dohrenwend, B. P. (1978). Exemplification of method for scaling life events: The PERI life events scale. *Journal of Health and Social Behavior*, 19, 205–229.
- Egan, G. (2013). *The skilled helper: a problem-management and opportunity-development approach to helping* (10th ed.). Pacific Grove, CA: Brooks/Cole.
- Erdberg, P., & Exner, J. E., Jr. (1984). Rorschach assessment. In G. Goldstein & M. Hersen (Eds.), *Handbook of Psychological Assessment*. New York: Pergamon Press.
- Exner, J. E. (1978). *The Rorschach: A comprehensive system: Volume 2. Current research and advanced interpretation*. New York: John Wiley & Sons.
- Exner, J. E. (1986). *The Rorschach: A comprehensive system: Volume 1. Basic foundations* (2nd ed.). New York: John Wiley & Sons.
- Fabrega, H., Jr. (2002). Evolutionary theory, culture and psychiatric diagnosis. In M. Maj & W. Gaebel (Eds.), *Psychiatric diagnosis and classification* (pp. 107–135). New York: John Wiley & Sons.
- Febbraro, G. A. R., & Clum, G. A. (1998). Meta-analytic investigation of the effectiveness of self-regulatory components in the treatment of adult behavior problems. *Clinical Psychology Review*, 18, 143–161.
- Feldman Barrett, L. (2003). So you want to be a social neuroscientist? *APS Observer*, 16, 5–7.
- Flinn, L., Braham, L., & Nair, R. D. (2014). How reliable are case formulations? A systematic literature review. *British Journal of Clinical Psychology*. Advance online publication. doi: 10.1111/bjc.12073
- Foa, E. B., Riggs, D. S., Marsie, E. D., & Yarczower, M. (1995). The impact of fear activation and anger on the efficacy of exposure treatment for posttraumatic stress disorder. *Behavior Therapy*, 26, 487–499.
- Gale, C. R., Batty, G. D., Tynelius, P., Deary, I. J., & Rasmussen, F. (2010). Intelligence in early adulthood and subsequent hospitalization for mental disorders. *Epidemiology*, 21, 70–77.
- Garb, H. N. (2005). Clinical judgment and decision making. *Annual Review of Clinical Psychology*, 1, 67–89.
- Gaw, A. C. (2001). *Concise guide to cross-cultural psychiatry*. Washington, DC: American Psychiatric Association.
- Ghanem, K. G., Hutton, H. E., Zenilman, J. M., Zimba, R., & Erbeling, E. J. (2005). Audio computer assisted self interview and face to face interview modes in assessing response bias among STD clinic patients. *Sexually Transmitted Infections*, 81(5), 421–425.
- Glass, C. R., & Arnkoff, D. B. (1997). Questionnaire methods of cognitive self-statement assessment. *Journal of Consulting and Clinical Psychology*, 65, 911–927.
- Golden, C. J. (1981a). The Luria-Nebraska Children's Battery: Theory and formulation. In G. W. Hynd & J. E. Obrzut (Eds.), *Neuropsychological assessment and the school-age child: Issues and procedures*. New York: Grune & Stratton.
- Golden, C. J. (1981b). A standardized version of Luria's neuropsychological tests: A quantitative and qualitative approach to neuropsychological evaluation. In S. B. Filskov & T. J. Boil (Eds.), *Handbook of clinical neuropsychology*. New York: John Wiley & Sons.
- Golden, C. J., Hammeke, T., & Purisch, A. (1978). Diagnostic validity of a standardized neuropsychological battery derived from Luria's neuropsychological tests. *Journal of Consulting and Clinical Psychology*, 46, 1258–1265.
- Graham, J. (2011). *MMPI-2: Assessing personality and psychopathology*, (5th ed.). New York: Oxford University Press.
- Hanson, R. K., Hunsley, J., & Parker, K. C. (1988). The relationship between WAIS subtest reliability, "g" loadings, and meta-analytically derived validity estimates. *Journal of Clinical Psychology*, 44(4), 557–563.
- Harvey, A. G., & Bryant, R. A. (2002). Acute stress disorder: A synthesis and critique. *Psychological Bulletin*, 128, 886–902.
- Harvey, A. G., Watkins, E., Mansell, W., & Shafran, R. (2004). *Cognitive behavioural processes across psychological disorders: A transdiagnostic approach to research and treatment*. Oxford, UK: Oxford University Press.
- Hathaway, S. R., & McKinley, J. C. (1943). *MMPI manual*. New York: Psychological Corporation.
- Haynes, S. N., & Horn, W. F. (1982). Reactivity in behavioral observation: A review. *Behavioral Assessment*, 4, 369–385.
- Horton, A. M. J. (2008). The Halstead-Reitan Neuropsychological Test Battery: Past, present, and future. In A. M. Horton & D. Wedding (Eds.), *The Neuropsychology Handbook* (3rd ed.) (pp. 251–278). New York: Springer Publishing.
- Hunsley, J., & Bailey, J. M. (1999). The clinical utility of the Rorschach: Unfulfilled promises and an uncertain future. *Psychological Assessment*, 11, 266–277.
- Hurlburt, R. T. (1979). Random sampling of cognitions and behavior. *Journal of Research on Personality*, 13, 103–111.
- Hurlburt, R. T. (1997). Randomly sampling thinking in the natural environment'. *Journal of Consulting and Clinical Psychology*, 65(6), 941–949.
- Hyman, S. E. (2002). Neuroscience, genetics, and the future of psychiatric diagnosis. *Psychopathology*, 35, 139–144.
- Hyman, S. E. (2010). The diagnosis of mental disorders: The problem of reification. *Annual Review of Clinical Psychology*, 6, 155–179.
- Insel, T. R. (2014). The NIMH Research Domain Criteria (RDoC) Project: Precision Medicine for Psychiatry. *American Journal of Psychiatry*, 171(4), 395–397.

- Jablensky, A., Sartorius, N., Ernberg, G., Anker, M., Korten, A., Cooper, J. E., ... Bertelsen A. (1992). Schizophrenia: manifestations, incidence and course in different cultures. A World Health Organization Ten Country Study. *Psychological Medicine*. Monograph Supplement 20. Cambridge: Cambridge University Press.
- Kashden, J., & Franzen, M. D. (1996). An interrater reliability study of the Luria-Nebraska Neuropsychological Battery Form-II quantitative scoring system. *Archives of Clinical Neuropsychology*, 11, 155–163.
- Kendler, K. S., Hettema, J. M., Butera, F., Gardner, C. O., & Prescott, C. A. (2003). Life event dimensions of loss, humiliation, entrapment, and danger in the prediction of onsets of major depression and generalized anxiety. *Archives of General Psychiatry*, 60, 789–796.
- Kendler, K. S., Jacobson, K. C., Prescott, C. A., & Neale, M. C. (2003). Specificity of genetic and environmental risk factors for use and abuse/dependence of *Cannabis*, cocaine, hallucinogens, sedatives, stimulants, and opiates in male twins. *American Journal of Psychiatry*, 160, 687–695.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, 62, 593–602.
- Kraemer, H., Kupfer, D. J., Clarke, D. E., Narrow, W. E., & Regier, D. A. (2012). DSM-5: How reliable is reliable enough? *American Journal of Psychiatry*, 169, 13–15.
- Krueger, R. F., Markon, K. E., Patrick, C. J., & Iacono, W. G. (2005). Externalizing psychopathology in adulthood: A dimensional-spectrum conceptualization and its implications for DSM-V. *Journal of Abnormal Psychology*, 114, 537–550.
- Leigh, A., & Gong, X. (2009). Estimating cognitive gaps between indigenous and non-indigenous Australians. *Education Economics*, 17, 239–261. doi: 10.1080/09645290802069418
- Lewis-Fernandez, R., Gorritz, M., Raggio, G. A., Pelaez, C., Chen, H., & Guarnaccia, P. J. (2010). Association of trauma-related disorders and dissociation with four idioms of distress among Latino psychiatric outpatients. *Cultural and Medical Psychiatry*, 34(2), 219–243.
- Liddell, B. J., Nickerson, A., Sartor, L., Ivancic, L., & Bryant, R. A. (2016). The generational gap: Mental disorder prevalence and disability amongst first and second generation immigrants in Australia. *Journal of Psychiatric Research*, 83, 103–111. doi: 10.1016/j.jpsychires.2016.08.011
- Lilienfeld, S. O., Lynn, S. J., Ruscio, J., & Beyerstein, B. L. (2010). Sad, mad, and bad: Myths about mental illness. In S. O. Lilienfeld, S. J. Lynn, J. Ruscio & B. L. Beyerstein (Eds.), *50 great myths of popular psychology: Shattering widespread misconceptions about human behavior* (pp. 181–208). Hoboken, NJ: John Wiley & Sons.
- Lilienfeld, S. O., Wood, J. M., & Garb, H. N. (2000). The scientific status of projective techniques. *Psychological Science in the Public Interest*, 1, 27–66.
- Lim, D., Sanderson, K., & Andrews, G. (2000). Lost productivity among full-time workers with mental disorders. *Journal of Mental Health Policy and Economics*, 3, 139–146.
- Lopez, S. R. (1989). Patient variable biases in clinical judgment: Conceptual overview and methodological considerations. *Psychological Bulletin*, 106, 184–203.
- Lopez, S. R. (1994). Latinos and the expression of psychopathology: A call for direct assessment of cultural influences. In C. Telles & M. Karno (Eds.), *Latino mental health: Current research and policy perspectives*. Los Angeles: UCLA.
- Lopez, S. R. (1996). Testing ethnic minority children. In B. B. Wolman (Ed.), *The encyclopedia of psychology, psychiatry, and psychoanalysis*. New York: Holt.
- Lopez, S. R. (2002). Teaching culturally informed psychological assessment: Conceptual issues and demonstrations. *Journal of Personality Assessment*, 79, 226–234.
- Lopez-Ibor, J. J., Jr. (2003). Cultural adaptations of current psychiatric classifications: Are they the solution? *Psychopathology*, 36, 114–119.
- Macneil, C. A., Hasty, M. K., Conus, P., & Berk, M. (2012). Is diagnosis enough to guide interventions in mental health? Using case formulation in clinical practice. *BMC Medicine*, 10, 111.
- Matthewson, M., Langworthy, J., & Higgins, D. (2015). Psychological predictors of vocational success for people with psychotic illness. *Australian Journal of Rehabilitation Counselling*, 21, 29–64. doi: 10.1017/jrc.2015.2
- McFall, R. M., & Hammen, C. L. (1971). Motivation, structure, and self-monitoring: Role of nonspecific factors in smoking reduction. *Journal of Consulting and Clinical Psychology*, 37, 80–86.
- McKown, C., & Weinstein, R. S. (2003). The development and consequences of stereotype consciousness in middle childhood. *Child Development*, 74, 498–515.
- McQuaid, J. R., Monroe, S. M., Roberts, J. R., & Johnson, S. L., et al. (1992). Toward the standardization of life stress assessment: Definitional discrepancies and inconsistencies in methods. *Stress Medicine*, 8, 47–56.
- Meyer, G. J., & Archer, R. P. (2001). The hard science of Rorschach research: “What do we know and where do we go?” *Psychological Assessment*, 13, 486–502.
- Moses, J. A., & Purisch, A. D. (1997). The evolution of the Luria-Nebraska Battery. In G. Goldstein & T. Incagnoli (Eds.), *Contemporary approaches to neuropsychological assessment* (pp. 131–170). New York: Plenum.
- Moses, J. A., Schefft, B. A., Wong, J. L., & Berg, R. A. (1992). Interrater reliability analyses of the Luria-Nebraska neuropsychological battery, form II. *Archives of Clinical Neurology*, 7, 251–269.
- Narrow, W. E., Clarke, D. E., Kuramoto, S. J., Kraemer, H. C., Kupfer, D. J., Greiner, L., & Regier, D. A. (2013). DSM-5 field trials in the United States and Canada, Part III: development and reliability testing of a cross-cutting symptom assessment for DSM-5. *American Journal of Psychiatry*, 170(1), 71–82.

- Nelson, R. O., Lipinski, D. P., & Black, J. L. (1976). The reactivity of adult retardates' self-monitoring: A comparison among behaviors of different valences, and a comparison with token reinforcement. *Psychological Record*, 26, 189–201.
- NHMRC (2007). National statement on ethical conduct in human research (2007) — Updated December 2013 (*the National Statement*). Commonwealth of Australia, Canberra.
- O'Brien, W. H., & Haynes, S. N. (1995). Behavioral assessment. In L. A. Heiden & M. Hersen (Eds.), *Introduction to clinical psychology* (pp. 103–139). New York: Plenum.
- Oakley Browne, M. A., Wells, J. E., & Scott, K. M., (Eds). (2006). *Te Rau Hinengaro: The New Zealand Mental Health Survey*. Wellington: Ministry of Health.
- Parker, G., Cheah, Y. C., & Roy, K. (2001). Do the Chinese somatize depression? A cross-cultural study. *Social Psychiatry Psychiatric Epidemiology*, 36, 287–293.
- Przeworski, A., & Newman, M. G. (2006). Efficacy and utility of computer-assisted cognitive behavioural therapy for anxiety disorders. *The Clinical Psychologist*, 10, 43–53.
- Regier, D. A., Narrow, W. E., Clarke, D. E., Kraemer, H. C., Kuramoto, S. J., Kuhl, E. A., & Kupfer, D. J. (2013). DSM-5 field trials in the United States and Canada, Part II: Test-retest reliability of selected categorical diagnoses. *American Journal of Psychiatry*, 170, 59–70.
- Reynolds, C. R., Chastain, R. L., Kaufman, A. S., & McLean, J. E. (1997). Demographic characteristics and IQ among adults: Analysis of the WAIS-R standardization sample as a function of the stratification variables. *Journal of School Psychology*, 25, 323–342.
- Rogler, L. H., & Hollingshead, A. B. (1985). *Trapped: Families and schizophrenia* (3rd ed.). Maplewood, NJ: Waterfront Press.
- Rorschach, H. (1921). *Psychodiagnostik: methodik und ergebnisse eines wahrnehmungsdiagnostischen experiments*. Berlin: Dritte aufl. Hrsg. von W. Morgenthaler.
- SANE Australia. (2013). *A life without stigma: A SANE Report*. SANE Australia.
- Sartorius, N., Jablensky, A., Korten, A., Ernberg, G., et al. (1986). Early manifestations and first-contact incidence of schizophrenia in different cultures: A preliminary report on the initial evaluation phase of the WHO Collaborative Study on Determinants of Outcome of Severe Mental Disorders. *Psychological Medicine*, 16, 909–928.
- Selye, H. (1950). *The physiology and pathology of exposure to stress*. Montreal: Acta.
- Spencer, S. J., Steele, C. M., & Quinn, D. M. (1999). Stereotype threat and women's math performance. *Journal of Experimental Social Psychology*, 35, 4–28.
- Spitzer, R. L., Gibbon, M., & Williams, J. B. W. (1996). *Structured clinical interview of DSM-IV Axis I disorders*. New York: N. Y. State Psychiatric Institute, Biometrics Research Department.
- Stone, A. A., & Shiffman, S. (1994). Ecological momentary assessment (EMA) in behavioral medicine. *Annals of Behavioral Medicine*, 16, 199–202.
- Stone, A. A., Schwartz, J., Neale, J. M., Shiffman, S., Marco, C. A., et al. (1998). A comparison of coping assessed by ecological momentary assessment and retrospective recall. *Journal of Personality and Social Psychology*, 74, 1670–1680.
- Sue, S., Yan Cheng, J. K., Saad, C. S., & Chu, J. P. (2012). Asian American mental health: A call to action. *American Psychologist*, 67(7), 532–544.
- Suzuki, K., Takei, N., Kawai, M., Minabe, Y., & Mori, N. (2003). Is taijin kyofusho a culture-bound syndrome? *American Journal of Psychiatry*, 160(7), 1358.
- Sweet, J. J., Carr, M. A., Rossini, E., & Kasper, C. (1986). Relationship between the Luria-Nebraska Neuropsychological Battery and the WISC-R: Further examination using Kaufman's factors. *International Journal of Clinical Neuropsychology*, 8, 177–180.
- Trierweiler, S. J., Neighbors, H. W., Munday, C., Thompson, E. E., Binion, V. J., & Gomez, J. P. (2000). Clinician attributions associated with the diagnosis of schizophrenia in African American and non-African American patients. *Journal of Consulting and Clinical Psychology*, 68, 171–175.
- Tsai, D. C., & Pike, P. L. (2000). Effects of acculturation on the MMPI-2 scores of Asian American students. *Journal of Personality Assessment*, 74, 216–230.
- Tsai, J. L. (2014). [personal communication].
- Tsai, J. L., Butcher, J. N., Vitousek, K., & Munoz, R. (2001). Culture, ethnicity, and psychopathology. In H.E. Adams & P.B. Sutker (Eds.). *The Comprehensive Handbook of Psychopathology* (pp. 105–127). New York, NY: Plenum Press.
- Turner, C. F., Ku, S. M., Rogers, L. D., Lindberg, J. H., & Pleck, F. L. (1998). Adolescent sexual behavior, drug use, and violence: Increased reporting with computer survey technology. *Science*, 280, 867–873.
- Van Ameringen, M. A., Lane, R. M., Walker, J. R., et al. (2001). Sertraline treatment of generalized social phobia: A 20-week, double-blind, placebo-controlled study. *American Journal of Psychiatry*, 158, 275–281.
- Walters, G. L., & Clopton, J. R. (2000). Effect of symptom information and validity scale information on the malingering of depression on the MMPI-2. *Journal of Personality Assessment*, 75, 183–199.
- Watson, D. (2005). Rethinking the mood and anxiety of disorders: A qualitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, 114, 522–536.
- Watters, E. (2010). *Crazy like us: The globalization of the America psyche*. New York: Free Press.
- Weissman, A., & Beck, A. T. (1978). *Development and validation of the Dysfunctional Attitude Scale: A preliminary investigation*.

- Whiteford, H. A., Ferrari, A. J., Baxter, A. J., Charlson, F. J., Degenhardt, L. (2014). How did we arrive at burden of disease estimates for mental and illicit drug use disorders in the Global Burden of Disease Study 2010? *Current Opinion in Psychiatry*, 26, 376–383. doi: 10.1097/YCO.0b013e328361e60f
- WHO World Mental Health Survey Consortium. (2004). Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization world mental health surveys. *Journal of the American Medical Association*, 291, 2581–2590.
- World Health Organization. (2001). *World health report: New understanding, new hope*. Geneva: Author.
- Zimmerman, M., & Mattia, J. I. (1999). Differences between clinical and research practices in diagnosing borderline personality disorder. *American Journal of Psychiatry*, 156, 1570–1574.

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## CHAPTER 3

# Mood disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 3.1** describe the symptoms of depression and mania, the diagnostic criteria for depressive disorders and bipolar disorders, and the epidemiology of these disorders
  - 3.2** discuss the genetic, neurobiological, social and psychological factors that contribute to the mood disorders
  - 3.3** identify the medication and psychological treatments of mood disorders, as well as the current views of electroconvulsive therapy
  - 3.4** explain the epidemiology and risk factors associated with suicide, as well as methods for preventing suicide.
-

## OPENING SCENARIO

Mary M, a 38-year-old mother of four children, had been deeply depressed for about two months when she first went to see a psychologist. Three years earlier, she had returned to work when the cost of sending their children to a private school made it hard for her family to get by on her husband's income as a high school teacher. About seven months before her visit to the psychologist, she had lost her job as an administrative assistant, which was a serious blow to the family's finances. She felt guilty about the loss of her job and became preoccupied with signs of her overall incompetence. Each night, she struggled for more than an hour to fall asleep, only to wake up frequently throughout the night. She had little appetite and as a result had lost five kilograms. She also had little energy for and no interest in activities that she had enjoyed in the past. Household chores became impossible for her to do and her husband began to complain. Their marriage had already been strained for two years, and her negativity and lack of energy contributed to further arguments. Finally, realising that Mary's symptoms were serious, Mr M talked her into making an appointment with a GP, who then referred Mary to a psychologist. (You will read about the outcome of Mary's treatment later in this chapter.)

### QUESTIONS

1. What questions might Mary's psychologist need to ask her to clarify possible diagnoses?
2. Losing her job represents a significant loss for Mary. Given this loss, would her psychologist consider this depression or a normal grief reaction? Why or why not?

## Introduction

Mood disorders involve disabling disturbances in emotion — from the extreme sadness and disengagement of depression to the extreme elation and irritability of mania. In this chapter, we begin by discussing the clinical description and the epidemiology of the different mood disorders. Next, we consider various perspectives on the aetiology of these disorders and then we consider approaches to treating them. We conclude with an examination of suicide, an action far too often associated with mood disorders.

## 3.1 Clinical descriptions and epidemiology of mood disorders

**LEARNING OUTCOME 3.1** Describe the symptoms of depression and mania, the diagnostic criteria for depressive disorders and bipolar disorders, and the epidemiology of these disorders.

The DSM-5 recognises two broad types of **mood disorders**: those that involve only depressive symptoms and those that involve manic symptoms (bipolar disorders). Table 3.1 presents a summary of the symptoms of each of these disorders. We will begin by discussing the depressive disorders and then we will turn to the bipolar disorders. Within each, we will describe the core signs, the formal criteria for the specific disorders and then the epidemiology and consequences of these disorders. After covering the clinical description and epidemiology of depressive and bipolar diagnoses, we describe the DSM-5 specifiers (subtypes) that are used to further define depressive disorders and bipolar disorders.

### Depressive disorders

The cardinal symptoms of depression include profound sadness and/or an inability to experience pleasure. Most of us experience sadness during our lives and most of us say that we are 'depressed' at one time or another. But most of these experiences do not have the intensity and duration to be diagnosable. The author William Styron (1992) wrote about his depression: 'Like anyone else I have always had times when I felt deeply depressed, but this was something altogether new in my experience — a despairing, unchanging paralysis of the spirit beyond anything I had ever known or imagined could exist.'

The symptoms of depression are varied. When people develop a depressive disorder, their heads may reverberate with self-recriminations. Like Mary, described in the opening scenario, they may become focused on their flaws and deficits. Paying attention can be so exhausting that they have difficulty absorbing what they read and hear. They often view things in a very negative light and they tend to lose hope. Initiative may disappear. Social withdrawal is common; many prefer to sit alone and be silent. Some people with depression neglect their appearance. When people become utterly dejected and hopeless, thoughts about suicide are common.

Physical symptoms of depression are also common, including fatigue and low energy as well as physical aches and pains. These symptoms can be profound enough to convince afflicted persons that they must be suffering from some serious medical condition, even though the symptoms have no apparent physical cause (Simon, Von Korff, Piccinelli, Fullerton, & Ormel, 1999). Although people with depression typically feel exhausted, they may find it hard to fall asleep and may wake up frequently. Other people sleep throughout the day. They may find that food tastes bland or that their appetite is gone, or they may experience an increase in appetite. Sexual interest disappears. Some may find their limbs feel heavy. Thoughts and movements may slow for some (**psychomotor retardation**), but others cannot sit still — they pace, fidget and wring their hands (**psychomotor agitation**).

#### DSM-5

##### DSM-5 criteria for major depressive disorder

Sad mood or loss of pleasure in usual activities are a symptom of major depressive disorder.

Additional criteria includes experiencing at least five of the following symptoms (counting sad mood and loss of pleasure):

- sleeping too much or too little
- psychomotor retardation or agitation
- weight loss or change in appetite
- loss of energy
- feelings of worthlessness or excessive guilt
- difficulty concentrating, thinking or making decisions
- recurrent thoughts of death or suicide.

Symptoms are present nearly every day, most of the day, for at least two weeks. Symptoms are distinct and more severe than a normative response to significant loss.

**TABLE 3.1** Overview of the major DSM-5 mood disorders

DSM-5 diagnoses	Major features
Major depressive disorder	Five or more depressive symptoms, including sad mood or loss of pleasure, for two weeks
Persistent depressive disorder	Low mood and at least two other symptoms of depression at least half of the time for two years
Premenstrual dysphoric disorder	Mood symptoms in the week before menses
Disruptive mood dysregulation disorder	Severe recurrent temper outbursts and persistent negative mood for at least one year beginning before age 10
Bipolar I disorder	At least one lifetime manic episode
Bipolar II disorder	At least one lifetime hypomanic episode and one major depressive episode
Cyclothymia	Recurrent mood changes from high to low for at least two years, without hypomanic or depressive episodes

## Major depressive disorder

The DSM-5 diagnosis of **major depressive disorder (MDD)** requires five depressive symptoms to be present for at least two weeks. These symptoms must include either depressed mood or loss of interest and pleasure. As shown in the DSM-5 criteria, additional symptoms must be present, such as changes in sleep, appetite, concentration or decision making, feelings of worthlessness, suicidality, or psychomotor agitation or retardation.

MDD is an **episodic disorder**, because symptoms tend to be present for a period of time and then clear. Even though episodes tend to dissipate over time, an untreated episode may stretch on for five months or even longer. Some people improve enough that they no longer meet the criteria for diagnosis of MDD but continue to experience subclinical depression for years (Judd et al., 1998).

Major depressive episodes tend to recur — once a given episode clears, a person is likely to experience another episode. About two-thirds of people with an episode of major depression will experience at least one more episode during their lifetime (Solomon et al., 2000). The average number of episodes is about four (Judd, 1997). With every new episode that a person experiences, his or her risk for experiencing another episode goes up by 16 percent (Solomon et al., 2000).

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Some people with depression have trouble falling asleep and staying asleep. Others find themselves sleeping for more than 10 hours but still feeling exhausted.



## Persistent depressive disorder

People with **persistent depressive disorder** are chronically depressed — more than half of the time for at least two years, they feel blue or derive little pleasure from usual activities and pastimes. In addition, they have at least two of the other symptoms of depression. The central feature of this diagnosis is the chronicity of symptoms, which has been shown to be a stronger predictor of poor outcome than the number of symptoms. Among people who have experienced depressive symptoms for at least two years, those who do and do not have a history of major depressive disorder appear similar in their symptoms and treatment response (McCullough et al., 2000). Persistent depressive disorder is similar to a DSM-IV-TR diagnosis of dysthymia.

## DSM-5

### DSM-5 criteria for persistent depressive disorder (dysthymia)

The criteria for persistent depressive disorder includes having a depressed mood for most of the day more than half of the time for two years (or one year for children and adolescents).

Individuals will experience at least two of the following symptoms during that time:

- poor appetite or overeating
- sleeping too much or too little
- low energy
- poor self-esteem
- trouble concentrating or making decisions
- feelings of hopelessness.

Persistent depressive disorder can be diagnosed if symptoms do not clear for more than two months at a time and bipolar disorders are not present.

Australian comedian Magda Szubanski has written in her autobiography *Reckoning* about her long battle with depression. One out of every five women will experience an episode of depression during her lifetime.



### Gender differences in depression

Women are twice as likely as men are to experience major depression and persistent depressive disorder and the gender ratio is particularly pronounced in countries and cultural groups with more traditional gender roles (Seedat et al., 2009). For example, this gender difference does not hold between Jewish adults, because depression is more common among Jewish men than among other men (Levav, Kohn, Golding, & Weissman, 1997). In Australia, the 12-month prevalence of depressive episodes is 5.1 percent for women, as compared with 3.1 percent for men (ABS, 2008). Some of you might wonder if these findings just reflect a tendency for men to be less likely to describe symptoms. So far, evidence does not support that idea (Kessler, 2003). Although a fair amount of research has focused on hormonal factors that could explain the vulnerability of women, findings have been mixed for this idea, too (Brems, 1995). Several social and psychological factors may help explain this gender difference (Nolen-Hoeksema, 2001).

- Twice as many girls as boys are exposed to childhood sexual abuse.
- During adulthood, women are more likely than men to be exposed to chronic stressors such as poverty and caretaker responsibilities.
- Acceptance of traditional social roles among girls may intensify self-critical attitudes about appearance. Adolescent girls worry more than adolescent boys about their body image, a factor that appears tied to depression (Hankin & Abramson, 2001).
- Traditional social roles may interfere with pursuit of some potentially rewarding activities that are not considered 'feminine'.
- Exposure to childhood and chronic stressors, as well as the effects of female hormones, could change the reactivity of the HPA axis, a biological system guiding reactions to stress.
- A focus on gaining approval and closeness within interpersonal relationships, which is more commonly endorsed by women, may intensify reactions to interpersonal stressors (Hankin, Mermelstein, & Roesch, 2007).
- Social roles promote emotion-focused coping among women, which may then extend the duration of sad moods after major stressors. More specifically, women tend to spend more time ruminating about sad moods or wondering about why unhappy events have occurred. Men tend to spend more time using distracting or action-focused coping, such as playing a sport or engaging in other activities that shake off the sad mood. As we discuss when we review cognitive factors in depression later in this chapter, a fair amount of research suggests that rumination will intensify and prolong sad moods.

In all likelihood, gender differences in depression are related to multiple factors. In considering these issues, bear in mind that men are more likely to demonstrate other types of disorders, such as alcohol and substance abuse as well as antisocial personality disorder (Seedat et al., 2009). Hence, understanding gender differences in psychopathology is likely to require attending to many different risk factors and syndromes.

#### QUESTIONS

1. What might be some reasons that gender differences in depression are less pronounced in societies with less traditional gender roles?
2. What aspects of Australian and New Zealand culture could contribute to or reduce gender differences in depression?

### Other DSM-5 depressive disorders

The DSM-5 includes two depressive disorders that were not listed as mood disorders in the DSM-IV-TR. First, **disruptive mood dysregulation disorder** is a newly defined depressive disorder. Second, **premenstrual dysphoric disorder** has been moved from the DSM-IV appendix on conditions

for further study to the main diagnostic section. We briefly discuss the rationale for disruptive mood dysregulation, a diagnosis specific to children and adolescents, in a later chapter. Because so little is known, we do not discuss these two diagnoses further.

## Epidemiology and consequences of depressive disorders

MDD is one of the most common psychological disorders. The Australian Bureau of Statistics (ABS) reports a 12-month prevalence of depressive episodes at 4.1 percent (ABS, 2008). New Zealand population surveys report similar prevalence (5.7 percent) (Wells, 2006). Although prevalence estimates are not yet available for the DSM-5 diagnosis of persistent depressive disorder, we do know that chronic forms of depression are rarer than MDD: the 12-month prevalence of dysthymia as defined by DSM-IV was 1.3 percent in Australia and 1.1 percent in New Zealand.

MDD and persistent depressive disorder are both twice as common among women as among men (Seedat et al., 2009; see focus on discovery 3.1 for a discussion of possible reasons for this gender difference). Socioeconomic status also matters — that is, MDD is three times as common among people who are impoverished compared to those who are not (Kessler, Berglund, Borges, Nock, & Wang, 2005).

The prevalence of depression varies considerably across cultures. In a major cross-cultural study using the same diagnostic criteria and structured interview in each country, prevalence of MDD varied from a low of 6.5 percent in China to a high of 21 percent in France (Bromet et al., 2011). It is tempting to assume that differences in prevalence rates by country indicate a strong role for culture. It turns out that differences between countries in rates of depression may be fairly complex. As described in focus on discovery 3.2, one factor may be distance from the equator. Rates of winter depression, or seasonal affective disorder, are higher farther from the equator, where days are shorter. There is also a robust correlation of per-capita fish consumption with depression; countries with higher fish consumption, such as Japan and Iceland, have much lower rates of MDD and bipolar disorder (Hibbeln, Nieminen, Blasbalg, Riggs, & Lands, 2006). Undoubtedly, cultural and economic factors, such as family cohesion and wealth disparity, play an important role in rates of depression as well.

The symptom profile of a depressive episode also varies across cultures and one likely reason is the differences in cultural standards regarding acceptable expressions of emotional distress. For example, people in South Korea are less likely to describe a sad mood or suicidal thoughts than are people in the United States (Chang et al., 2008). Complaints of nerves and headaches are common in Latino culture and reports of weakness, fatigue and poor concentration are common in some Asian cultures. On the other hand, these symptom differences do not appear to be major enough to explain the differing rates of depression across countries (Simon, Goldberg, Von Korff, & Ustun, 2002), particularly when careful studies are conducted.

### FOCUS ON DISCOVERY 3.2

#### Seasonal affective disorder: the winter blues

Criteria for the seasonal specifier of MDD specify that a person experiences depression during two consecutive winters and that the symptoms clear during the summer. These winter depressions appear to be much more common in northern than in southern climates; for example, in the US, while less than 2 percent of people living in sunny Florida report these patterns, about 10 percent of people living in northern New Hampshire report seasonal affective disorder (Rosen et al., 1990). The prevalence of seasonal affective disorder in Australia is thought to be lower, with a Melbourne study producing an estimate of 0.3 percent (Murray, 2004).

For mammals living in the wild, a slower metabolism in the winter could have been a lifesaver during periods of scarce food. For some unlucky humans, though, this same mechanism might contribute to **seasonal affective disorder**. It is believed that seasonal affective disorder is related to changes in the levels of melatonin in the brain. Melatonin is exquisitely sensitive to light and dark cycles and is only released during dark periods. People with seasonal affective disorder show greater changes in melatonin in the winter than do people without seasonal affective disorder (Wehr et al., 2001). Factors other than melatonin also are likely to contribute to seasonal mood shifts; for example, people with seasonal affective disorder show a change in their retinal sensitivity to light that appears to be genetically guided (Roeklein et al., 2013).

Hibernation in animals typically involves sleeping long hours, along with lowered appetite and energy. Indeed, the most common human response to seasonal shifts is sleep, appetite and energy changes. Some people, though, seem to respond to those physical changes self-critically (Young, Watel, Lahmeyer, & Eastman, 1991). For example, faced with a lack of energy to tackle new projects, some people may feel guilty. People prone to self-criticism and other negative cognitions may be particularly likely to develop the full constellation of depressive symptoms in the face of the seasonal shifts in energy, sleep and appetite.

Fortunately, several treatment options are available for seasonal affective disorder. Like other subtypes of depression, seasonal affective disorder responds to antidepressant medications and cognitive-behavioural therapy (Rohan et al., 2007). Winter blues, though, are as likely to remit with 30 minutes of bright light each morning as with fluoxetine (Prozac) (Lam et al., 2006). At least eight high-quality studies have examined bright light as a treatment for seasonal affective disorder (Golden et al., 2005) and it is established as a first-line recommendation in the American Psychiatric Association treatment guidelines for depression. Intriguingly, light therapy has been shown to help relieve depression even among those without a seasonal pattern to their depressions (Lieveise et al., 2011).

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This woman is having light therapy, which is an effective treatment for patients with seasonal depression.



Seasonal affective disorder is most common in regions far from the equator.



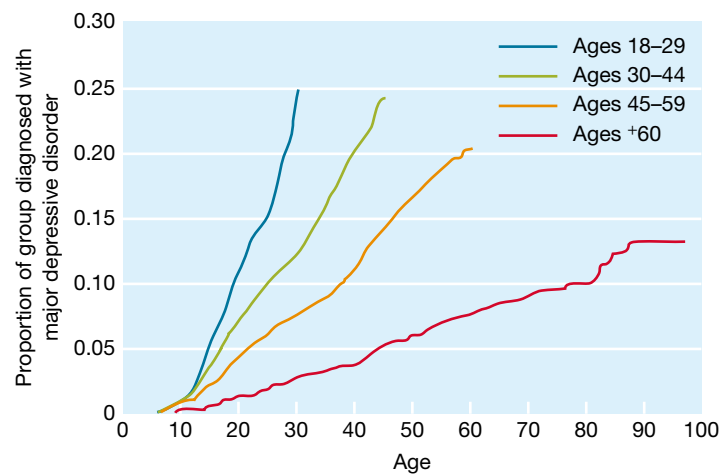
### QUESTIONS

1. What behavioural symptoms of depression are likely to compound seasonal impacts on mood?
2. Why might the prevalence of seasonal affective disorder be lower in Australia as compared to the northern hemisphere?

In most countries, the prevalence of MDD increased steadily during the mid- to late twentieth century (Klerman, 1988); at the same time, the age of onset decreased (Kessler, Birnbaum et al., 2010). Figure 3.1 shows that the age of onset has become lower for each recent generation of people in the United States: among people in their 60s, less than 5 percent reported that they had experienced an episode of MDD by age 20, whereas among people aged 18–29, almost 10 percent reported that they had experienced an episode of MDD by age 20. The median age of onset is now the late teens to early 20s, and depression is now all too present in universities. One possible explanation for the increasing depression rates lies in the social changes that have occurred over the past 100 years. Support structures — such as tightly knit extended families and marital stability, which were more widespread in the past — are often absent for people today. Yet there are no clear data about why depression is striking earlier and earlier.

Both MDD and persistent depressive disorder are often associated, or comorbid, with other psychological disorders. About 60 percent of people who meet the criteria for diagnosis of MDD during their lifetime also will meet the criteria for diagnosis of an anxiety disorder at some point (Kessler et al., 2003). See focus on discovery 3.4 later in the chapter for more discussion of the overlap of anxiety disorders and depressive disorders. Other common comorbid conditions include substance-related disorders, sexual dysfunctions and personality disorders.

**FIGURE 3.1** With each generation, the median age of onset for major depressive disorder gets younger.



*Source:* Adapted from Kessler, Berglund, Demler et al. (2003).

Depression has many serious consequences. From the depths of a depression, getting to work may be far too effortful, parenting can feel like a burden and suicide can seem like an option. As we will discuss later in this chapter, suicide is a real risk. MDD is also one of the world's leading causes of disability (Murray & Lopez, 1996); it is estimated to be the fourth leading cause of non-fatal disease burden in Australia (Australian Institute of Health and Welfare, 2016) and it is estimated that MDD is associated with \$12.6 billion per year in lost productivity and job turnover in Australia (LaMontagne, 2010). MDD also has important implications for the next generation: offspring who are exposed to their mother's MDD during early childhood are at high risk for developing depression (Hammen, Hazel, Brennan, & Najman, 2012).

MDD is also related to a high risk of other health problems, including death from medical diseases (Mykletun et al., 2009). There is particularly strong evidence that depression is related to the onset and more severe course of cardiovascular disease. Across 22 prospective studies, depression has been found to predict a 60 percent increase in the severity of cardiovascular disease over time (Nicholson, Kuper, & Hemingway, 2006). Depression is also related to increased risk of death from cardiovascular disease, even after controlling for baseline cardiovascular health (Barth, Schumacher, & Herrmann-Lingen, 2004).

Although the diagnostic criteria for persistent depressive disorder require fewer symptoms than for MDD, do not make the mistake of thinking that persistent depressive disorder is a less severe disorder than MDD. One study found that these chronic depressive symptoms persisted on average for more than five years and that functioning declined as the symptoms persisted for more years (Klein, Shankman, & Rose, 2006). The chronicity of these symptoms takes a toll. Indeed, a study following patients for five years found that people with chronic depressive symptoms were more likely to require hospitalisation, to attempt suicide and to be impaired in their functioning than were people with MDD (Klein, Schwartz, Rose, & Leader, 2000).

## Bipolar disorders

The DSM-5 recognises three forms of bipolar disorders: bipolar I disorder, bipolar II disorder and cyclothymic disorder. Manic symptoms are the defining feature of each of these disorders. The bipolar disorders are differentiated by how severe and long-lasting the manic symptoms are.

These disorders are labelled ‘bipolar’ because most people who experience mania will also experience depression during their lifetime (mania and depression are considered opposite poles). Contrary to what people may believe, an episode of depression is not required for a diagnosis of bipolar I (although two-thirds to three-fourths of people with an episode of mania will experience an episode of depression; see Cuellar, Johnson, & Winters, 2005). Depression is required for a diagnosis of bipolar II disorder.

**Mania** is a state of intense elation, irritability or activation accompanied by other symptoms shown in the diagnostic criteria. During manic episodes, people will act and think in ways that are highly unusual compared to their typical selves. They may become louder and make an incessant stream of remarks, sometimes full of puns, jokes, rhymes and interjections about nearby stimuli that have attracted their attention (like Wayne in the following clinical case). They may be difficult to interrupt and may shift rapidly from topic to topic, reflecting an underlying **flight of ideas**. During mania, people may become sociable to the point of intrusiveness. They can also become excessively self-confident. They may stop sleeping but remain incredibly energetic. Attempts by others to curb such excesses can quickly bring anger and even rage. Mania often comes on suddenly over a period of a day or two. For many, the boundless energy, bursts of joy and incredible surge in goals can seem welcome, so that some fail to recognise the sudden changes as a sign of disorder. The following passage from Kay Redfield Jamison illustrates some of the rapid shifts that can occur.

When you’re high it’s tremendous. The ideas and feelings are fast and frequent like shooting stars and you follow them until you find better and brighter ones. Shyness goes, the right words and gestures are suddenly there, the power to captivate others a felt certainty. There are interests found in uninteresting people. Sensuality is pervasive and the desire to seduce and be seduced irresistible. Feelings of ease, intensity, power, well-being, financial omnipotence and euphoria pervade one’s marrow. But, somewhere, this changes. The fast ideas are far too fast and there are far too many; overwhelming confusion replaces clarity. Memory goes. Humor and absorption on friends’ faces are replaced by fear and concern. (Jamison, 1993, pp. 67–68)

Unfortunately, people can be oblivious to the potentially disastrous consequences of their manic behaviour, which can include risky sexual activities, overspending and reckless driving. The following quote, also from Kay Redfield Jamison, illustrates some of the impulsivity of this state.

When I am high I couldn’t worry about money if I tried. So I don’t. The money will come from somewhere; I am entitled; God will provide . . . So I bought twelve snakebite kits, with a sense of urgency and importance. I bought precious stones, elegant and unnecessary furniture, three watches within an

American comedian Maria Bamford created a semi-autobiographical comedy TV series about her experiences with bipolar disorder.



hour of one another (in the Rolex rather than Timex class: champagne tastes bubble to the surface, are the surface, in mania) and totally inappropriate siren-like clothes. During one spree in London I spent several hundred pounds on books having titles or covers that somehow caught my fancy: books on the natural history of the mole, twenty sundry Penguin books because I thought it could be nice if the penguins could form a colony. Once I think I shoplifted a blouse because I could not wait a minute longer for the woman-with-molasses feet in front of me in line. Or maybe I just thought about shoplifting, I don't remember, I was totally confused. I imagine I must have spent far more than thirty thousand dollars during my two major manic episodes and God only knows how much more during my frequent milder manias. But then back on lithium and rotating on the planet at the same pace as everyone else, you find your credit is decimated, your mortification complete. (Jamison, 1993, p. 75)

The DSM-5 also includes criteria for **hypomania** (see diagnostic criteria for manic and hypomanic episodes). *Hypo-* comes from the Greek for 'under'; hypomania is 'under' — less extreme than — mania. Although mania involves significant impairment, hypomania does not. Rather, hypomania involves a change in functioning that does not cause serious problems. The person with hypomania may feel more social, energised, productive and sexually alluring.

#### CLINICAL CASE

##### Wayne

Wayne, a 32-year-old claims adjuster, had been married for 8 years. He lived happily with his wife and their two children in a middle-class neighbourhood. He had not experienced any clear symptoms until one morning when he told his wife that he was bursting with energy and ideas, that his job was unfulfilling and that he was just wasting his talent. That night he slept little, spending most of the time at his desk, writing furiously. The next morning he left for work at the usual time but returned home at 11.00 am, his car overflowing with aquariums and other equipment for tropical fish. He had quit his job, then withdrawn all the money from the family's savings account and spent it on tropical fish equipment. Wayne reported that the previous night he had worked out a way to modify existing equipment so that fish 'won't die anymore. We'll be millionaires'. After unloading the paraphernalia, Wayne set off to canvass the neighbourhood for possible buyers, going door to door and talking to anyone who would listen. Wayne reported that no one in his family had been treated for bipolar disorder, but his mother had gone through periods when she would stop sleeping and become extremely adventurous. For the most part, the family had regarded these episodes as unproblematic, with the exception of a period in which she had set off across the country without the children and had returned only after spending a major amount of money.

The following exchange indicates Wayne's incorrigible optimism:

**Therapist:** Well, you seem pretty happy today.

**Wayne:** Happy! Happy! You certainly are a master of understatement, you rogue! [Shouting, literally jumping out of his seat.] Why, I'm ecstatic! I'm leaving for Sydney today, on my daughter's bicycle. Only 700 kilometres. That's nothing, you know. I could probably walk, but I want to get there by next week. And along the way I plan to contact a lot of people about investing in my fish equipment. I'll get to know more people that way... Oh, God, how good it feels.

#### QUESTIONS

1. Are Wayne's symptoms suggestive of mania or hypomania?
2. What information would Wayne's therapist need to obtain to clarify which bipolar subtype Wayne is presenting with?

### Bipolar I disorder

In the DSM-5, the criteria for diagnosis of **bipolar I disorder** (formerly known as manic-depressive disorder) include a single episode of mania during the course of a person's life. Note, then, that a person who is

diagnosed with bipolar I disorder may or may not be experiencing current symptoms of mania. In fact, even someone who experienced only one week of manic symptoms years ago is still diagnosed with bipolar I disorder. Even more than episodes of MDD, bipolar episodes tend to recur. More than half of people with bipolar I disorder experience four or more episodes during their lifetimes (Goodwin & Jamison, 2007).

### Bipolar II disorder

The DSM-5 also includes a milder form of bipolar disorder, called **bipolar II disorder**. To be diagnosed with bipolar II disorder, a person must have experienced at least one major depressive episode and at least one episode of hypomania (and no lifetime episode of mania).

### Cyclothymic disorder

Also called *cyclothymia*, **cyclothymic disorder** is a second chronic mood disorder (the other is persistent depressive disorder). As with the diagnosis of persistent depressive disorder, the DSM-5 criteria require that symptoms be present for at least two years among adults. In cyclothymic disorder, the person has frequent but mild symptoms of depression, alternating with mild symptoms of mania. Although the symptoms do not reach the severity of full-blown hypomanic or depressive episodes, people with the disorder and those close to them typically notice the ups and downs.

Oscar-nominated New Zealand actress Keisha Castle-Hughes has discussed her diagnosis of bipolar disorder on social media. As is common, she was initially diagnosed with depression.



#### DSM-5

##### DSM-5 criteria for cyclothymic disorder

For at least two years (or one year in children or adolescents):

- numerous periods with hypomanic symptoms that do not meet criteria for a hypomanic episode
- numerous periods with depressive symptoms that do not meet criteria for a major depressive episode

The symptoms do not clear for more than two months at a time. Criteria for a major depressive, manic or hypomanic episode have never been met. Symptoms cause significant distress or functional impairment.

#### DSM-5

##### DSM-5 criteria for manic and hypomanic episodes

Criteria for manic and hypomanic episodes includes experiencing an elevated or irritable mood.

The person displays increased activity or energy, and at least three of the following are noticeably changed from baseline (four, if mood is irritable):

- increase in goal-directed activity or psychomotor agitation
- unusual talkativeness; rapid speech

- flight of ideas or subjective impression that thoughts are racing
- decreased need for sleep
- increased self-esteem; belief that one has special talents, powers or abilities
- distractibility; attention easily diverted
- excessive involvement in activities that are likely to have painful consequences, such as reckless spending, sexual indiscretions or unwise business investments
- symptoms are present most of the day, nearly every day.

For a manic episode:

- symptoms last one week, require hospitalisation or include psychosis
- symptoms cause significant distress or functional impairment.

For a hypomanic episode:

- symptoms last at least four days
- clear changes in functioning are observable to others, but impairment is not marked
- no psychotic symptoms are present.

## RESEARCH EXAMPLE

### Bipolar II disorder – the ‘milder’ disorder?

Hypomania in bipolar II disorder (in contrast to mania in bipolar I disorder) is said to not cause significant problems, leading to bipolar II disorder being thought of as a ‘milder’ form of the condition. An Australian study (Fletcher, Parker, Paterson, & Synnott, 2013) explored risk-taking behaviours and their impacts in bipolar II disorder. Ninety-three people with a diagnosis of bipolar II disorder completed a questionnaire detailing behaviours undertaken while hypomanic.

The results indicated that the majority of participants had engaged in risky behaviours during hypomania, such as overspending (including purchasing cars and real estate), dangerous driving, substance abuse, thrill-seeking and risky sexual activity. These behaviours had significant potential for harm, including consequences to relationships, physical health and financial stability.

Despite the often enjoyable highs of hypomania and possible benefits such as creativity (see focus on discovery 3.3), the study shows hypomania carries the potential for significant harm for sufferers, reinforcing the need for clinicians and their patients to consider treatment and risk-management plans. The study also serves as a reminder that despite the DSM-5 using severity to distinguish bipolar II from bipolar I disorder, bipolar II is by no means minimally or non-impairing.

### QUESTION

List the key distinguishing characteristics between bipolar I disorder and bipolar II disorder.

## Epidemiology and consequences of bipolar disorders

Bipolar I disorder is much rarer than MDD. In an epidemiological study that involved structured diagnostic interviews with a representative sample of 61 392 people across 11 countries, about 6 out of 1000 (0.6 percent) people met the criteria for bipolar I disorder (Merikangas et al., 2011).

It is hard to estimate the prevalence of milder forms of bipolar disorder because some of the most commonly used diagnostic interviews are not reliable. When researchers conducted interrater reliability tests of bipolar II disorder using structured clinical interviews, the diagnosis was confirmed for less than half of the people tested (Kessler et al., 2006). (Many of those were diagnosed with other forms of bipolar disorder.) As you might expect given the low reliability, the prevalence estimates vary, with large-scale epidemiological studies suggesting that bipolar II disorder affects somewhere between 0.4 percent and 2 percent of people (Merikangas et al., 2007, 2011). It is estimated that about 4 percent of people experience cyclothymic disorder (Regeer et al., 2004). In Australia and New Zealand, large-scale studies have produced estimates of the 12-month prevalence of any bipolar disorder that range between 1.8 and 2.2 percent (ABS, 2008; Wells, 2006).

More than half of those with bipolar spectrum disorders report onset before age 25 (Merikangas et al., 2011), but like depression, bipolar disorders are being seen with increasing frequency among children and adolescents (Kessler, Berglund, Demler et al., 2005). Although bipolar disorders occur equally often in men and women, women diagnosed with bipolar disorder experience more episodes of depression than do men with this diagnosis (Altshuler et al., 2010). About two-thirds of people diagnosed with bipolar disorder meet diagnostic criteria for a comorbid anxiety disorder and many report a history of substance abuse.

Bipolar I disorder is among the most severe forms of psychological disorders. One-third of people remain unemployed a full year after hospitalisation for mania (Harrow, Goldberg, Grossman, & Meltzer, 1990). Suicide rates are high for both bipolar I and bipolar II disorders (Angst, Stassen, Clayton, & Angst, 2002). People with these disorders are at high risk for a range of other medical conditions, including cardiovascular disease, diabetes, obesity and thyroid disease (Kupfer, 2005) and these problems are often quite severe. People who have been hospitalised for bipolar I disorder are twice as likely to die from medical illnesses in a given year as are people without mood disorders (Osby, Brandt, Correia, Ekblom, & Sparen, 2001). These sad consequences of bipolar disorders are not offset by evidence that bipolar individuals and their family members possess heightened creativity or are high achievers (see focus on discovery 3.3).

### FOCUS ON DISCOVERY 3.3

#### Creativity and mood disorders

In her book *Touched with Fire: Manic-Depressive Illness and the Artistic Temperament* (1993), Kay Redfield Jamison, an expert on bipolar disorders and herself a long-time sufferer from bipolar I disorder, assembled evidence linking mood disorders, especially bipolar disorder, to artistic creativity. Of course, most people with mood disorders are not exceptionally creative, and most creative people do not have mood disorders — but the list of visual artists, composers and writers who seem to have experienced mood disorders is impressive. The list includes Michelangelo, van Gogh, Tchaikovsky, Schumann, Gauguin, Tennyson, Shelley, Faulkner, Hemingway, F Scott Fitzgerald and Whitman. In recent years, many actors and actresses have also spoken out about their history of mania, including Stephen Fry, Carrie Fisher and Linda Hamilton. The biographical findings dovetail with results from a systematic review that suggests that individuals with bipolar disorder and their unaffected family members are more likely to choose creative occupations and have their creative accomplishments recognised by others (Johnson, Edge, Holmes, & Carver, 2012).

Many people assume that the manic state itself fosters creativity through elated mood, increased energy, rapid thoughts and a heightened ability to make connections among seemingly unrelated events. Extreme mania, however, lowers creative output and even if people produce more work during a manic period, the quality of that work might suffer, as seems to have been the case for the composer Robert Schumann (Weisberg, 1994). Moreover, studies have shown that people who have experienced episodes of mania tend to be less creative than those who have had the milder episodes

Mood disorders are common among artists and writers. Tchaikovsky was affected. Frank Sinatra is quoted as having said this about himself: 'Being an 18-karat manic depressive and having lived a life of violent emotional contradictions, I have an over-acute capacity for sadness as well as elation.'



of hypomania and both groups tend to produce less creative output than do non-ill family members (Richards, Kinney, Lunde, Benet, & Merzel, 1988). Although many people with bipolar disorder worry that taking medications may limit their creativity, these findings suggest that reducing manic symptoms should help, rather than hurt, creativity.

### QUESTIONS

1. How might states of hypomania promote creativity and what aspects of extreme mania are likely to lower creative output?
2. What are the implications of studies that show enhanced creativity in unaffected family members of individuals with bipolar disorder?

People with cyclothymia are at elevated risk for developing episodes of mania and major depression. Even if full-blown manic episodes do not emerge, the chronicity of cyclothymic symptoms takes its toll.

## Subtypes of depressive disorders and bipolar disorders

The mood disorders are highly heterogeneous — that is, people who have been diagnosed with the same disorder may show very different symptoms. The DSM-5 addresses this by providing criteria for dividing MDD and bipolar disorders into a number of specifiers (subtypes). See table 3.2 for a list of specifiers and their definitions. Both the **rapid cycling** specifier (see figure 3.2) and the seasonal specifier refer to the pattern of episodes over time, whereas other specifiers refer to the specific symptoms of the current episode. All of the specifiers can be applied to either MDD or bipolar disorders, with the exception of rapid cycling, which is diagnosed only for bipolar disorder. All of the episode specifiers can be applied to either depressed or manic episodes, with the exception of **melancholic**, which is only relevant for episodes of depression.

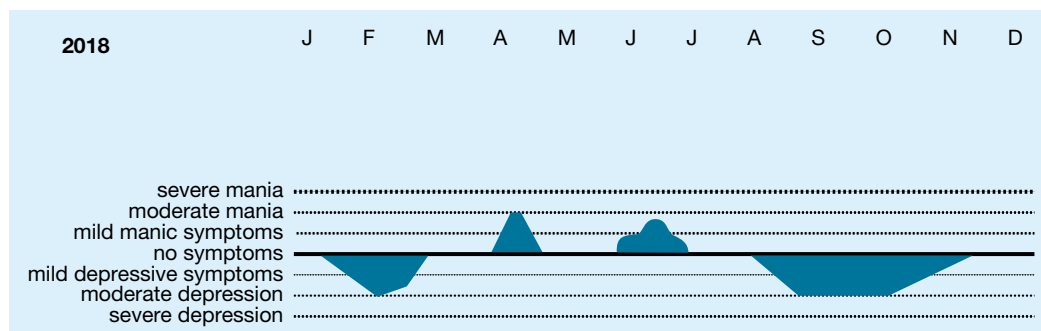
**TABLE 3.2** Specifiers of depressive disorders and bipolar disorders

Subtype	Applicable to MDD?	Applicable to bipolar disorder?	Definition
Seasonal pattern	Yes	Yes	Episodes happen regularly at a particular time of the year
Rapid cycling	No	Yes	At least four episodes within the past year
Mood-congruent psychotic features	Yes	Yes	Delusions or hallucinations with themes that are consistent with the mood state (e.g., guilt, disease or death themes accompanying depression)
Mood-incongruent psychotic features	Yes	Yes	Delusions or hallucinations with themes that do not match the valence of the depressive or manic episode
Mixed features	Yes	Yes	At least three manic symptoms are present during a depressive episode, or at least three depressive symptoms are present during a manic episode
Catatonia	Yes	Yes	Extreme physical immobility or excessive peculiar physical movement
Melancholic features	Yes	Yes (for depressive episodes)	Lack of pleasure in any activity, inability to gain relief from positive events and at least three other symptoms of depression, such as a distinct quality of mood, depressive symptoms that are worse in the morning, waking at least two hours too early, loss of appetite/weight, psychomotor retardation or agitation, or guilt

Subtype	Applicable to MDD?	Applicable to bipolar disorder?	Definition
Atypical features	Yes	Yes	Symptoms that are unusual for depressive or manic episodes are present
Peripartum onset	Yes	Yes Onset during pregnancy or within 4 weeks postpartum	
With anxious distress	Yes	Yes	At least two symptoms of anxiety are present
Suicide risk severity	Yes	Yes	Suicidal ideation, plans or other risk indicators are present

The seasonal specifier of MDD has achieved a fair amount of support (see focus on discovery 3.2 for discussion), but many of the other specifiers have not been well validated. For example, although about 14 percent of women meet criteria for the **peripartum onset** specifier of depression, depression is not much more common in the postpartum period compared to other periods in life and about a half of depressive episodes that are present during pregnancy or after birth actually started before the pregnancy (Wisner et al., 2013). Similarly, problems exist with the rapid cycling specifier. In a longitudinal study, 95 percent of persons with rapid cycling bipolar disorder at baseline were no longer experiencing rapid cycling one year later (Schneck et al., 2008). As a third example of problems with specifiers, MDD with melancholic features may just be a more severe type of depression — that is, people with melancholic features have more comorbidity (e.g., with anxiety disorders), more frequent episodes of depression and more impairment in everyday activities (Kendler, 1997).

**FIGURE 3.2** The rapid cycling specifier of bipolar disorder is defined by at least four mood episodes per year, as shown in this case.



## 3.2 Aetiology of mood disorders

**LEARNING OUTCOME 3.2** Discuss the genetic, neurobiological, social and psychological factors that contribute to the mood disorders.

When we think of the profound extremes embodied in the mood disorders, it is natural to ask why these extremes happen. How can we explain Mary sinking into the depths of depression? What factors combined to drive Wayne into his frenzied state of unrealistic ambitions? Studies of aetiology focus on

why these disorders unfold. No single cause can explain mood disorders. A number of different factors combine to explain their onset.

While the DSM includes several different depressive disorders and bipolar disorders, the research on aetiology and treatment has tended to focus on major depressive disorder and bipolar I disorder. For simplicity, we refer to these conditions as depression and bipolar disorder through the remainder of this chapter.

We begin by discussing biological factors involved in depression and bipolar disorder. As table 3.3 shows, many different biological approaches have been applied to mood disorders and we will describe genetic, neurotransmitter, brain-imaging and neuroendocrine research. After describing these biological risk factors for depressive and bipolar disorders, we discuss psychosocial predictors of depression and then turn to psychosocial models of bipolar disorder.

**TABLE 3.3 Summary of neurobiological hypotheses about major depression and bipolar disorder**

Neurobiological hypothesis	Major depression	Bipolar disorder
Genetic contribution	Moderate	High
Serotonin and dopamine receptor dysfunction	Present	Present
Cortisol dysregulation	Present	Present
Changes in activation of emotion-relevant regions in the brain	Present	Present
Activation of the striatum in response to reward	Low	High

## Genetic factors

The more careful studies of MZ (identical) and DZ (fraternal) twins yield heritability estimates of 37 percent for MDD (Sullivan, Neale, & Kendler, 2000). As described in chapter 1, heritability can be interpreted as the percent of the variance in depression that is explained by genes. Heritability estimates are higher when researchers study more severe samples (e.g., when the people in the study are recruited in inpatient hospitals rather than outpatient clinics).

Bipolar disorder is among the most heritable of disorders. The most careful studies involve interviews with representative samples of twins selected from the community (rather than focusing only on people who seek treatment, who may have more severe cases of the disorder than those who are not treated). One community-based twin sample that used structured interviews to verify diagnoses obtained a heritability estimate of 93 percent (Kieseppa, Partonen, Haukka, Kaprio, & Lonnqvist, 2004). Adoption studies also confirm the importance of heritability in bipolar disorder (Wender et al., 1986). Bipolar II disorder is also highly heritable (Edvardsen et al., 2008).

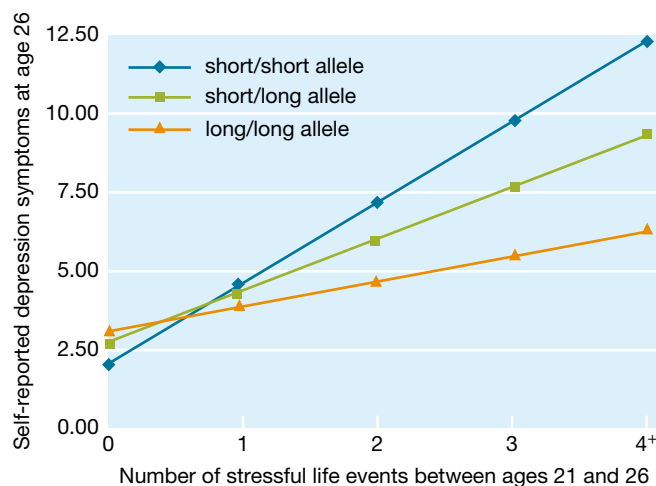
Molecular genetics research (see chapter 1) aims to identify the specific genes involved in mood disorders. To place molecular genetics research in context, it is worth pausing to consider the incredible range of depressive and bipolar symptoms that a person might experience. Because the mood disorders and indeed most psychological disorders, are so complex and heterogeneous, it is highly unlikely that there is one single gene that explains these illnesses. Most researchers believe that these disorders will be related eventually to a set of genes, with each gene accounting for a minute proportion of risk.

Likely due to the very small effects of any one gene, genome-wide association studies (GWAS) involving more than 18 000 persons have failed to identify specific genes associated with MDD (Sullivan, Daly, & O'Donovan, 2012) and many of the specific genes identified in smaller studies have failed to replicate (Krishnan & Nestler, 2010). In studies involving thousands of people, several genetic polymorphisms related to bipolar disorder have been identified. These polymorphisms explain a very

small proportion of the risk for bipolar disorder (Sullivan et al., 2012) and some of the genetic polymorphisms identified appear to overlap with those involved in schizophrenia (Cross-Disorder Group of the Psychiatric Genomics Consortium, CDGPGC, 2013).

A different approach is to consider whether a given gene might increase risk of depression in the presence of environmental risk factors. This approach shows that a polymorphism of the serotonin transporter gene does appear to be related to MDD. An initial study found that people with this polymorphism were at greater risk for depression after a stressful life event than those without the polymorphism (Caspi et al., 2003). That is, having at least one short allele was associated with elevated reactivity to stress (see figure 3.3). In rhesus monkeys, the presence of at least one short allele in this gene is associated with poor serotonergic function. Thus, some people seem to inherit a propensity for a weaker serotonin system, which is then expressed as a greater likelihood to experience depression after childhood maltreatment or an adulthood severe stressor. This finding has been replicated in other large-scale studies, particularly when the studies involved careful measurement of maltreatment and life stressors (Karg, 2011; Uher & McGuffin, 2010). Intriguingly, this polymorphism in the serotonin transporter gene has also been related to risk factors for depression that we will be discussing later in this chapter, including activity in key brain regions (e.g., the amygdala, described below; Caspi, Hariri, Holmes, Uher, & Moffitt, 2010) and negative cognitive tendencies (e.g., attention to negative information; Disner et al., 2013). This type of work, then, suggests that we should be considering genes for depression in concert with other risk factors.

**FIGURE 3.3** Life events interact with the serotonin transporter gene to predict symptoms of depression.

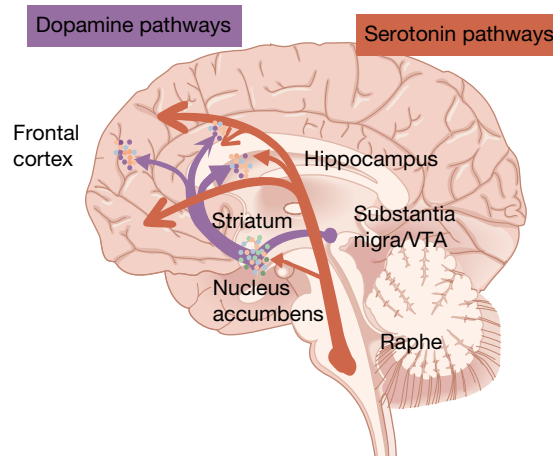


*Source:* Adapted from Caspi et al. (2003). *Science*, 301, 387.

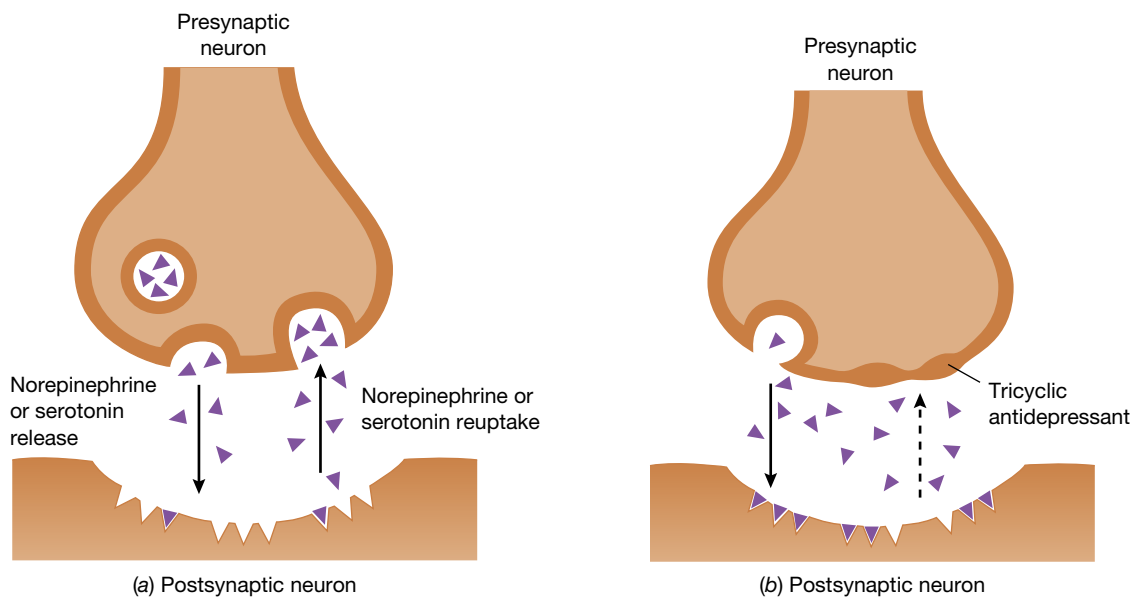
## Neurotransmitters

Three neurotransmitters have been studied the most in terms of their possible role in mood disorders: norepinephrine, dopamine and serotonin. Each of these neurotransmitters is present in many different areas of the brain. Figure 3.4 illustrates how widespread serotonin and dopamine pathways are in the brain. For decades, researchers studied the idea that mood disorders would be related to having too much or too little of a neurotransmitter, with mixed results. More recent models have focused on the idea that mood disorders might involve changes in receptors that respond to the presence of neurotransmitters in the synaptic cleft (see figure 3.5), with most of these newer studies focused on dopamine and serotonin.

**FIGURE 3.4** Serotonin and dopamine pathways are widespread in the brain.



**FIGURE 3.5** (a) When neurotransmitters are released into a synapse, a pump-like reuptake mechanism begins to recapture some of the neurotransmitter before it reaches the postsynaptic neuron. (b) Tricyclic drugs block this reuptake process so that more neurotransmitter reaches the receptor. Selective serotonin reuptake inhibitors act more selectively on serotonin.



*Source:* Adapted from Snyder (1996), p. 106.

How could one test the sensitivity of the postsynaptic receptors? If receptors are more or less sensitive, one might expect people to react differently to drugs that influence the level of a given neurotransmitter. For example, receptors that are overly sensitive may respond to even the smallest amount of a neurotransmitter in the synaptic cleft. If receptors are insensitive, a person may show a more pronounced response to a drop in neurotransmitter levels.

People with depression are less responsive than other people are to drugs that increase dopamine levels and it is thought that the functioning of dopamine might be lowered in depression (Naranjo, Tremblay, & Busto, 2001). Dopamine plays a major role in the sensitivity of the **reward system**

in the brain, which is believed to guide pleasure, motivation and energy in the context of opportunities to obtain rewards (Depue & Iacono, 1989). The diminished function of the dopamine system could help explain the deficits in pleasure, motivation and energy in depression (Treadway & Zald, 2011).

Among people with bipolar disorder, several different drugs that increase dopamine levels have been found to trigger manic symptoms. One possibility is that dopamine receptors may be overly sensitive in bipolar disorder (Anand et al., 2000), which might help explain the excessive energy and enthusiasm seen during manic episodes.

In addition to dopamine, studies have also focused on the sensitivity of serotonin receptors. Researchers have conducted a set of studies that involve experimentally lowering serotonin levels. To lower serotonin levels, researchers deplete levels of **tryptophan**, the major precursor of serotonin. Tryptophan can be depleted with a drink that contains high levels of 15 amino acids but no tryptophan. Within hours, serotonin levels are lowered, an effect that lasts for several hours. Studies show that depleting tryptophan (and so lowering serotonin levels) causes temporary depressive symptoms among people with a history of depression or a family history of depression (Benkelfat, Ellenbogen, Dean, Palmour, & Young, 1994; Neumeister et al., 2002). This effect is not observed among people with no personal or family history of depression. Current thinking is that people who are vulnerable to depression may have less sensitive serotonin receptors, causing them to respond more dramatically to lower levels of serotonin.

Like people diagnosed with MDD and their family members, relatives of those with bipolar disorder demonstrate stronger mood reactions to tryptophan depletion compared to matched controls (Sobczak, Honig, Nicolson, & Riedel, 2002). As with depression, it would appear that bipolar disorder may be related to diminished sensitivity of the serotonin receptors.

### Brain function: regions involved in emotion

Functional brain-imaging studies suggest that episodes of MDD are associated with changes in many of the brain systems that are involved in experiencing and regulating emotion (Davidson, Pizzagalli, & Nitschke, 2002). Many of these studies examine brain responses to emotional stimuli, such as pictures of very positive or very negative scenes. Table 3.4 summarises five primary brain structures that have been most studied in depression: the amygdala, the **anterior cingulate**, the **dorsolateral prefrontal cortex**, the hippocampus and the **striatum** (see also figure 3.6). We will discuss each of these regions, beginning with the amygdala.

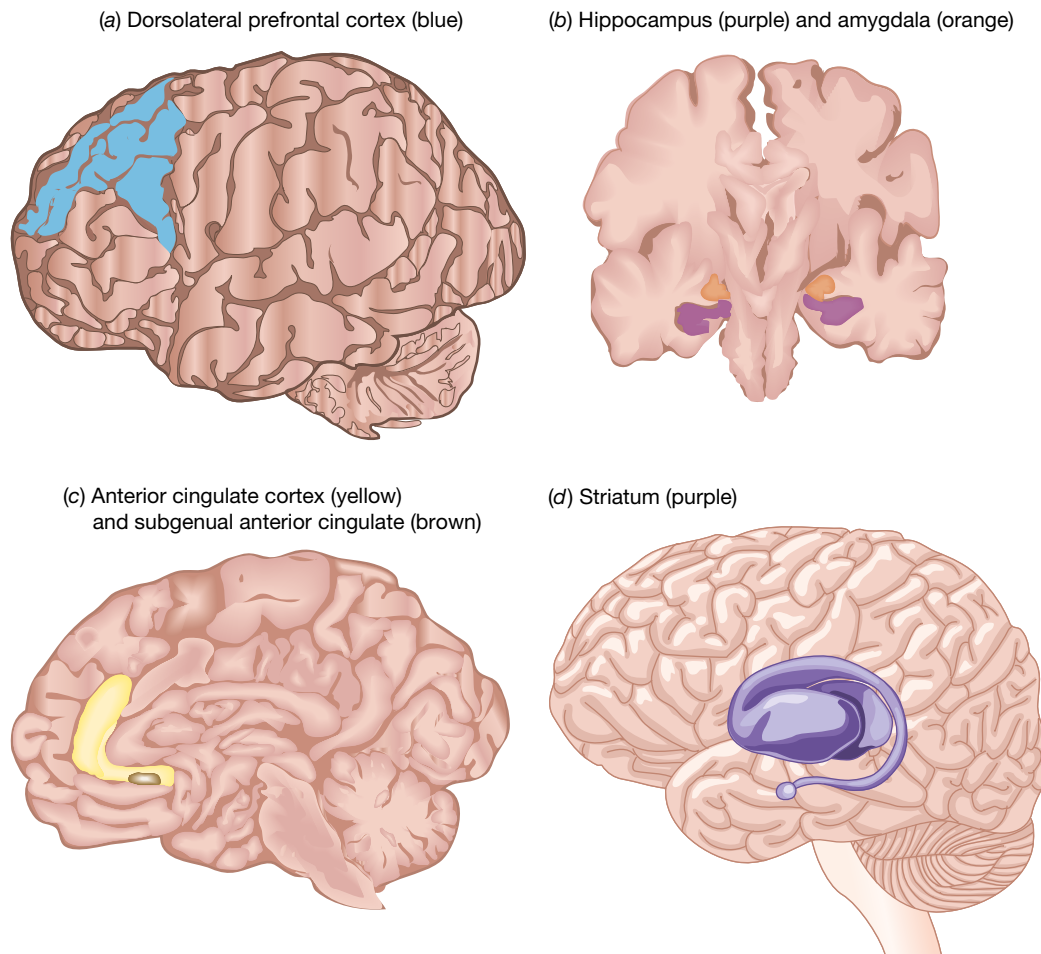
**TABLE 3.4** Activity of brain structures involved in emotion responses among people with mood disorders

Brain structure	Level in depression	Level in mania
Amygdala	Elevated	Elevated
Anterior cingulate	Elevated	Elevated
Dorsolateral prefrontal cortex	Diminished	Diminished
Hippocampus	Diminished	Diminished
Striatum	Diminished	Elevated

The amygdala helps assess how salient and emotionally important a stimulus is. For example, animals with damage to the amygdala fail to react with fear to threatening stimuli and also fail to respond positively to food. In humans, the amygdala has been shown to respond when people are shown pictures of threatening stimuli. Functional brain activation studies show elevated activity of the amygdala among people with MDD. For example, when shown negative words or pictures of sad or angry faces, people with current MDD have a more intense and sustained reaction in the amygdala than do people with no MDD (Sheline et al., 2001). This pattern of amygdala overreactivity to emotional stimuli can be shown

among relatives of people with depression who have no personal history of MDD, suggesting this might be part of the vulnerability to depression rather than just the aftermath of being depressed (Pilhatsch et al., 2014).

**FIGURE 3.6** Key brain regions involved in mood disorders.



**Source:** Adapted from Davidson, *Annual Review of Psychology*, 53, (2002). Copyright 2002 by Annual Reviews, [www.annualreviews.org](http://www.annualreviews.org).

In addition to the amygdala, MDD is associated with greater activation of the anterior cingulate (Hamilton et al., 2012) and diminished activation of the hippocampus and dorsolateral prefrontal cortex when viewing negative stimuli (Hamilton et al., 2012; Schaefer, Putnam, Benca, & Davidson, 2006). Disturbances in these regions are believed to interfere with effective emotion regulation.

Finally, people with depression demonstrate diminished activation of the striatum during exposure to emotional stimuli (Hamilton et al., 2012), particularly when they are receiving positive feedback that they have earned a reward (Pizzagalli, Goetz, Ostacher, Iosifescu, & Perlis, 2008). A specific region of the striatum (called the nucleus accumbens) is a central component of the reward system in the brain and plays a key role in motivation to pursue rewards (Salamone & Correa, 2012). The lack of activity of the striatum in response to positive feedback, then, may help explain why people with depression feel less motivated by and less engaged in the positive events in their life.

How might these findings fit together? One theory is that the overactivity in the amygdala during depression causes oversensitivity to emotionally relevant stimuli. At the same time, systems involved in regulating emotions are compromised (the anterior cingulate, the hippocampus and the dorsolateral prefrontal cortex). Finally, neural regions involved in responding and mobilising to rewards may also be underactive.

Many of the brain structures implicated in MDD also appear to be involved in bipolar disorder. Bipolar I disorder is associated with elevated responsiveness in the amygdala, increased activity of the anterior cingulate during emotion regulation tasks and diminished activity of the hippocampus and dorsolateral prefrontal cortex (Houenou et al., 2011; Phillips, Ladouceur, & Drevets, 2008). Although these patterns parallel those observed among people with MDD, one difference emerges. People with bipolar disorder tend to show high activation of the striatum (Chen, Suckling, Lennox, Ooi, & Bullmore, 2011), in contrast with the low activity observed for those with MDD. High activation of the striatum may help explain increased levels of reward sensitivity in people with bipolar disorder.

### **The neuroendocrine system: cortisol dysregulation**

The HPA axis (hypothalamic–pituitary–adrenocortical axis; see figure 1.11) may be overly active during episodes of MDD, which is consistent with the idea that stress reactivity is an important part of depression. As described above, there is evidence that the amygdala is overly reactive among people with MDD and the amygdala sends signals that activate the HPA axis. The HPA axis triggers the release of cortisol, the main stress hormone. Cortisol is secreted at times of stress and increases the activity of the immune system to help the body prepare for threats.

Various findings link depression to high cortisol levels. For example, people with **Cushing's syndrome**, which causes oversecretion of cortisol, frequently experience depressive symptoms. A second line of research with animals has shown that when chemicals that trigger cortisol release are injected into the brain, many of the classic symptoms of depression are produced, including decreased interest in sex, decreased appetite and sleep disturbance (Gutman & Nemeroff, 2003). In animals and humans, then, too much cortisol seems to produce depressive symptoms.

Even among people who are depressed but do not have Cushing's syndrome, cortisol levels are often poorly regulated — that is, the system does not seem to respond well to biological signals to decrease cortisol levels. To test this idea, researchers generally administer an agent such as dexamethasone, which suppresses cortisol secretion over the course of the night in healthy individuals. The dexamethasone suppression test (dex/CRH) is an even more sensitive test of the HPA system in which researchers administer both dexamethasone and corticotrophin-releasing hormone (which increases cortisol levels). About 80 percent of people hospitalised for depression show poor regulation of the HPA system on the dex/CRH test, that is, an elevated release of cortisol compared to healthy controls (Heuser, Yassouridis, & Holsboer, 1994). These abnormal responses to the dex/CRH test, though, normalise when the depressive episode ends for most people. People who continue to show elevated cortisol responses to the dex/CRH test after recovery from a depressive episode are more likely to relapse within the next year (e.g., Aubry et al., 2007).

Although cortisol helps mobilise beneficial short-term stress responses, prolonged high levels of cortisol can cause harm to body systems. For example, long-term excesses of cortisol have been linked to damage to the hippocampus — studies have found smaller-than-normal hippocampus volume among people who have experienced depression for years (Videbech & Ravnkilde, 2004).

Like people with MDD, people with bipolar disorder fail to demonstrate the typical suppression of cortisol after the dex/CRH test. This suggests that bipolar disorder is also characterised by a poorly regulated cortisol system (Watson, Thompson, Ritchie, Ferrier, & Young, 2006). Like those with MDD, people with bipolar disorder who continue to show abnormal responses to cortisol challenge tests after their episode clears are at high risk for more episodes in the future (Vieta et al., 1999).

In sum, both bipolar disorder and MDD are characterised by problems in the regulation of cortisol levels. Dysregulation in cortisol levels also predicts a more severe course of illness for bipolar disorder and MDD.

## Social factors in depression: childhood adversity, life events and interpersonal difficulties

Interpersonal problems are very common for those with depression, but showing cause and effect takes much more than just documenting this correlation. Depressive symptoms could easily contribute to interpersonal difficulties as the person withdraws, begins to feel irritable and finds no joy when engaging with others, but these same factors may also indicate individuals at risk of developing depression. To show that interpersonal concerns are not just an effect of the depressive symptoms, longitudinal studies showing that an interpersonal factor is present before onset are extremely important. Fortunately, many large longitudinal studies are available and we will focus on interpersonal variables that have been shown to precede and predict the onset of depressive episodes, including childhood adversity, negative life events, lack of social support, family criticism and an excessive need for interpersonal reassurance.

Childhood adversity, such as early parental death, physical abuse or sexual abuse, increases the risk that later, in their adolescence or adulthood, the person will develop depression (Daley, Hammen, & Rao, 2000) and that the depressive symptoms will be chronic (Klein et al., 2009). One aspect of childhood adversity — child abuse — can set the stage for many of the other risk factors in depression, including a negative cognitive style (Lumley & Harkness, 2007), poorer marital relationship quality (DiLillo, Giuffre, Tremblay, & Peterson, 2001), increased rates of life stress (Harkness, Bagby, & Kennedy, 2012) and altered activity in brain regions involved in depression (Hanson et al., 2013). Child abuse, however, is associated with a number of mental illnesses, suggesting that it acts as a non-specific risk factor for psychopathology generally. Child abuse may therefore increase the risk for different disorders, whereas other factors may shape whether depression occurs.

Beyond childhood adversity, the role of more recent stressful life events in triggering episodes of depression is well established. Prospective studies have shown that life events often precede a depressive episode. Even with a prospective study, though, it remains possible that some life events are caused by early symptoms of depression that have not yet developed into a full-blown disorder. Remember the case of Mary, from the opening scenario at the beginning of this chapter, who developed symptoms after she lost her job. Maybe Mary lost her job because her trouble waking in the morning caused her to arrive at work late; disturbed sleep patterns can be an early sign of depression. Even when researchers exclude stressful life events that could have been caused by mild depressive symptoms, there is much evidence that stress can cause depression. In careful prospective studies, 42 to 67 percent of people report that they experienced a very serious life event that was not caused by symptoms in the year before their depression began. Common events include losing a job, a key friendship or a romantic relationship (Brown & Harris, 1989).

Certain types of life events, such as those involving interpersonal loss and humiliation, appear particularly likely to trigger depressive episodes (Kendler, Hettema, Butera, Gardner, & Prescott, 2003). As examples, the death of a close other or a break-up with one's partner both seem to triple the risk of a depressive episode within the next several months (Kendler et al., 2003). Animals also show depressive-type symptoms after loss.

If it is natural to become depressed after a loss, some have argued that we should not pathologise such a normative response by assigning a diagnostic label (Wakefield, 2011). Others argue that the depressions that do or do not unfold in response to interpersonal loss seem parallel in their symptoms, treatment response and other core dimensions, such that we should consider interpersonal loss as just one of the many pathways into MDD (Kendler, Myers, & Zisook, 2008). The DSM-5 takes this latter approach and so these episodes receive the same diagnostic label as those that do not follow an interpersonal life event. Regardless of the diagnostic guidelines, we would do well to remember that depressive symptoms may be a fairly common response to major losses. We discuss one model of the role of interpersonal loss and stress in provoking biological changes and depressive symptoms in focus on discovery 3.5.

Why do some people, but not others, become depressed after stressful life events? The obvious answer is that some people must be more vulnerable to stress than others. Disturbances in many of the neurobiological systems we described above could increase reactivity to stress. Neurobiological factors, then, may be diatheses (pre-existing vulnerabilities) that increase risk for mood disorders in the context of other triggers or stressors. Psychological and cognitive vulnerabilities also appear to be important. The most common models, then, consider both diatheses and stressors. Diatheses could be biological, social or psychological.

One diathesis may be a lack of social support. People who are depressed tend to have sparse social networks and to regard those networks as providing little support (Keltner & Kring, 1998). Low social support may lessen a person's ability to handle stressful life events. One study showed that women experiencing a severely stressful life event without support from a confidant had a 40 percent risk of developing depression, whereas those with a confidant's support had only a 4 percent risk (Brown & Andrews, 1986). Social support, then, seems to buffer against the effects of severe stressors.

Family problems are another important interpersonal predictor of depression. A long line of research has focused on **expressed emotion (EE)** — defined as a family member's critical or hostile comments towards or emotional overinvolvement with the person with depression. High EE strongly predicts relapse in depression. Indeed, one review of six studies found that 69.5 percent of patients in families with high EE relapsed within one year, compared to 30.5 percent of patient in families with low EE (Butzlaff & Hooley, 1998). In a community study, marital discord also predicted the onset of depression (Whisman & Bruce, 1999).

The onset of depression can also be predicted by certain types of problems in a person's interpersonal style. In one line of longitudinal research, an excessive need for reassurance has been found to predict depression. Among a group of undergraduates who were not initially depressed, those who described themselves as needing unusually high amounts of reassurance were more likely to develop depressive symptoms over a 10-week period (Joiner & Metalsky, 2001). Among primary school children, low social competence predicted the onset of depression (Cole, Martin, Powers, & Truglio, 1990); and among adolescents, poor interpersonal problem-solving skills predicted increases in depression (Davila, Hammen, Burge, Paley, & Daley, 1995).

Clearly, interpersonal problems can trigger the onset of depressive symptoms, but it is also important to consider the flip side of the coin. Once depressive symptoms emerge, they can create interpersonal problems — that is, depressive symptoms seem to elicit negative reactions from others (Coyne, 1976). For example, roommates of American college students with depression rated social contacts with them as less enjoyable and they reported feeling hostility towards them (Joiner, Alfano, & Metalsky, 1992). Other research has shown that people respond poorly to constant reassurance seeking (Joiner, 1995). Taken together, it is clear that interpersonal loss, isolation and relationship concerns can trigger depression, but it is good to be aware that the depression and the related vulnerability can also create challenges in interpersonal relationships.

## Psychological factors in depression

Many different psychological factors may play a role in depressive disorders. In this section, we discuss personality and cognitive factors that operate as diatheses to increase the risk of depression in the context of stress. Here again, we will focus on some of the longitudinal findings documenting that personality and cognitive variables predict increases in depressive symptoms over time.

### Neuroticism

Several longitudinal studies suggest that **neuroticism**, a personality trait that involves the tendency to experience frequent and intense negative affect, predicts the onset of depression (Jorm et al., 2000). A large study of twins suggests that neuroticism explains at least part of the genetic vulnerability to depression (Fanous, Prescott, & Kendler, 2004). As you would expect, neuroticism is also associated with anxiety (Kotov, Gamez, Schmidt, & Watson, 2010), an overlap we discuss in focus on discovery 3.4.

### Understanding the overlap in anxiety and depression

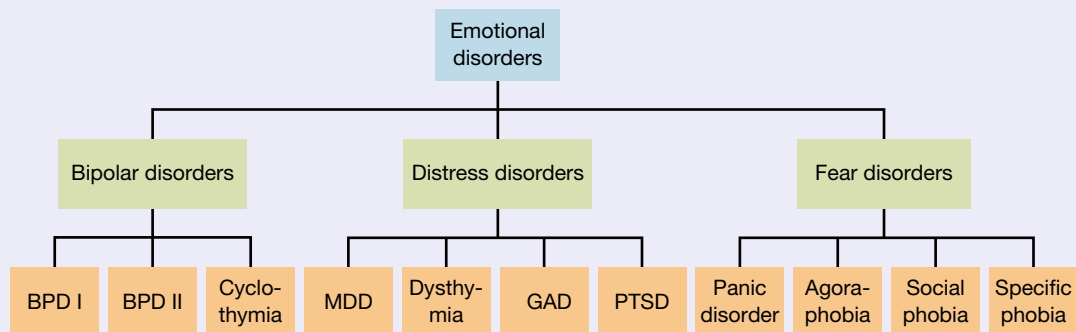
One idea proposed for DSM-5 was to consider whether anxiety disorders and depressive disorders are distinct. There are several reasons to question whether anxiety disorders should be considered to be separable from depressive disorders. Chief among these reasons is the high rate of comorbidity. At least 60 percent of people with an anxiety disorder will experience major depressive disorder during their lifetime and vice versa — about 60 percent of those with depression will experience an anxiety disorder (Kessler et al., 2003; Moffitt et al., 2007).

Certain anxiety disorders overlap with depressive disorders more than do others. Depression appears to be particularly likely to co-occur with generalised anxiety disorder (GAD; see a later chapter) and post-traumatic stress disorder (PTSD; see a later chapter) (Watson, 2009). Indeed, GAD is more correlated with depression than it is with other anxiety disorders (Watson, 2005). Beyond patterns of comorbidity, the aetiology of GAD, PTSD and depression overlaps. The genetic risk for GAD and depression overlaps substantially (Kendler, Jacobson, Prescott, & Neale, 2003). Major depression, persistent depressive disorder, GAD and PTSD each involve a propensity towards unhappiness and distress, whereas other anxiety disorders involve a propensity towards fear. Some risk factors seem tied to depression and to a range of anxiety disorders, such as neuroticism (Watson, 2005).

In response to this overlap, one group of researchers recommended that mood and anxiety disorders be subsumed into one larger chapter in the DSM-5 (Watson, O'Hara, & Stuart, 2008). Within that larger chapter, they recommended differentiating distress disorders: major depressive disorder, persistent depressive disorder, generalised anxiety disorder and PTSD; fear disorders: panic, agoraphobia, social phobia and specific phobia; and bipolar disorders (see figure 3.7). Although this categorisation was based on strong evidence from the epidemiological and genetic data, the crafters of DSM-5 decided not to make such a drastic change.

Rather, the DSM-5 includes the specifier 'with anxious distress' to be used when depressive episodes are accompanied by at least two anxiety symptoms. Many patients will likely meet the criteria for this specifier.

**FIGURE 3.7** A proposed revision of the emotional disorders



**Source:** Adapted from Watson, D. (2005). Rethinking the mood and anxiety disorder: A quantitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, 114(4), 522–536.

### QUESTIONS

1. Do you agree with the decision to categorise depressive disorders and anxiety disorders as distinct in the DSM-5? Why or why not?
2. What might be some reasons it is useful to think of anxiety disorders and depressive disorders as distinct, despite evidence of an overlap in prevalence and aetiology?

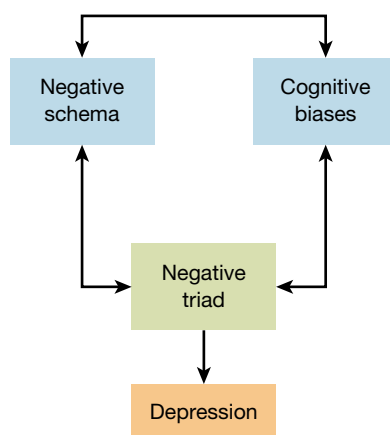
## Cognitive theories

Pessimistic and self-critical thoughts can torture the person with depression. In cognitive theories, these negative thoughts and beliefs are seen as major causes of depression. We will describe three cognitive theories. Beck's theory and hopelessness theory both emphasise these types of negative thoughts, although the theories differ in some important ways. Rumination theory emphasises the tendency to dwell on negative moods and thoughts. These models are not incompatible with the interpersonal and life stress research we discussed above; a person's negative thoughts may sometimes reflect genuinely stressful life circumstances. In cognitive models, though, the cognitions are seen as the most important force driving the depression.

### Beck's theory

Aaron Beck (1967) argued that depression is associated with a **negative triad**: negative views of the self, their world and the future (see figure 3.8). According to this model, in childhood, people with depression acquired negative schemas through experiences such as loss of a parent, the social rejection of peers or the depressive attitude of a parent. Schemas are different from conscious thoughts — they are an underlying set of beliefs that operate outside of a person's awareness to shape the way a person makes sense of his or her experiences. The negative schema is activated whenever the person encounters situations similar to those that originally caused the schema to form.

**FIGURE 3.8** The interrelationships among different kinds of cognitions in Beck's theory of depression



Once activated, negative schemas are believed to cause **cognitive biases**, or tendencies to process information in certain negative ways (Kendall & Ingram, 1989). That is, people with depression might attend to and remember even the smallest negative feedback about themselves, while at the same time failing to notice or remember positive feedback about themselves. People with a schema of ineptness might readily notice and remember signs that they are inept, while ignoring or forgetting signs that they are competent. Together, these cognitive errors lead them to make conclusions that are consistent with their underlying schema, which then maintains the schema (a vicious circle).

How has Beck's theory been tested? One widely used instrument in studies of Beck's theory is a self-report scale called the *Dysfunctional Attitudes Scale* (DAS), which includes items concerning whether people would consider themselves worthwhile or lovable. Hundreds of studies have shown that people demonstrate negative thinking on scales like the DAS during depression (Haaga, Dyck, & Ernst, 1991). Several major longitudinal studies suggest that people with negative cognitive styles are at elevated risk for developing depressive symptoms (Lewinsohn, Joiner, & Rohde, 2001), a first episode of MDD (Carter & Garber, 2011) and depressive relapse (Segal et al., 2006).

In studies of how people process information, depression is associated with a tendency to stay focused on negative information once it is initially noticed (Gotlib & Joormann, 2010). For example, if shown pictures of negative and positive facial expressions, those with depression tend to look at the negative pictures longer than they look at the positive pictures. People with depression also tend to remember more negative than positive information. In a meta-analysis of 25 studies, Mathews and MacLeod (2002) described evidence that most people who are not depressed will remember more positive than negative information. For example, if presented with a list of self-descriptive adjectives containing 20 negative and 20 positive words, most people will remember more of the positive than the negative words when queried later in a session. People with major depression, though, tend to remember about 10 percent more negative words than positive words. While non-depressed people seem to wear rose-coloured glasses, those with depression tend to have a negative bias in the way that they attend to and recall information.

As with self-reports of negative thoughts, cognitive biases in the way people process positive and negative information do seem to predict depression. In one study, 139 soldiers were tested before they went to the war in Iraq. Baseline tendencies to attend to sad faces were related to greater risk of developing depressive symptoms with exposure to the stresses of war (Beevers, Lee, Wells, Ellis, & Telch, 2011). Taken together, support has been obtained for Beck's model using a range of measures.

### Hopelessness theory

According to **hopelessness theory** (see figure 3.9; Abramson, Metalsky, & Alloy, 1989), the most important trigger of depression is hopelessness, which is defined by the belief that desirable outcomes will not occur and that there is nothing a person can do to change this. The model places emphasis on two key dimensions of **attributions** — the explanations a person forms about why a stressor has occurred (Weiner et al., 1971):

- stable (permanent) versus unstable (temporary) causes
- global (relevant to many life domains) versus specific (limited to one area) causes.

**FIGURE 3.9** Major elements of the hopelessness theory of depression

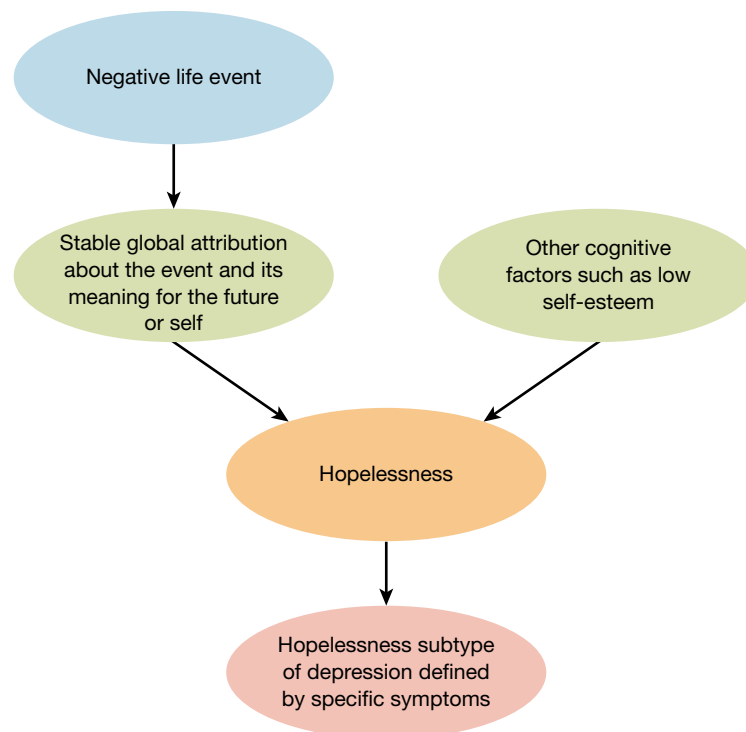


Table 3.5 illustrates these dimensions by considering how different people might explain their low score on a maths exam. People whose **attributional style** leads them to believe that negative life events are due to stable and global causes are likely to become hopeless and this hopelessness will set the stage for depression.

**TABLE 3.5** An example of attributions: why I failed my maths exam

	Stable	Unstable
Global	I lack intelligence.	I am exhausted.
Specific	I lack mathematical ability.	I am fed up with maths.

Gerald Metalsky and colleagues (1993) conducted the first test of hopelessness theory. Early in the semester, American college students completed the Attributional Style Questionnaire (ASQ), as well as questionnaires to assess their grade aspirations, depressive symptoms and hopelessness. These measures were used to predict depressive symptoms among students who received low grades on a test during the semester. Those who attributed poor grades to global and stable factors experienced more hopelessness and in turn, hopelessness predicted depressive symptoms. Clearly, these results support the hopelessness theory. A similar study conducted with children in the sixth and seventh grades yielded almost identical results (Robinson, Garber, & Hillsman, 1995).

One large-scale study has assessed several different aspects of cognitive theories of depression. In the Temple–Wisconsin Cognitive Vulnerability to Depression study, the DAS (a measure used to test Beck’s theory) and the ASQ (the measure used in tests of the hopelessness theory) were used to predict changes in depressive symptoms. Students with high scores on both the DAS and the ASQ were classified as high risk, whereas those with low scores on both measures were classified as low risk. Findings from this study provided support for cognitive theories: students in the high-risk group were more than six times as likely to develop first episodes of MDD in a two-year follow-up as were students in the low-risk group (Alloy, Abramson, Smith, Gibb, & Neeren, 2006).

### Rumination theory

While Beck’s theory and the hopelessness model tend to focus on the nature of negative thoughts, Susan Nolen-Hoeksema (1991) suggested that a way of thinking called **rumination** may increase the risk of depression. Rumination is defined as a tendency to repetitively dwell on sad experiences and thoughts, or to chew on material again and again. The most detrimental form of rumination may be a tendency to brood regretfully about why a sad event happened (Treynor, Gonzalez, & Nolen-Hoeksema, 2003).

Tendencies to ruminate, as measured using self-report scales, have been found to predict the onset of major depressive episodes among initially non-depressed persons (Just & Alloy, 1997; Morrow & Nolen-Hoeksema, 1990; Nolen-Hoeksema, 2000). As described in focus on discovery 3.1, one interesting aspect of this theory is that women tend to ruminate more than men do, perhaps because of sociocultural norms about emotion and emotion expression. The tendency for women to ruminate more may help explain the higher rates of depression among women as compared to men (Nolen-Hoeksema, 2000).

Dozens of experimental studies have been conducted to see how inducing rumination affects moods. Typically, in the rumination–induction condition, participants are exposed to stress and then asked to dwell on their current feelings and on themselves (e.g., ‘Think about the way you feel inside’), whereas in a distraction (control) condition, participants are asked to think about topics unrelated to their self or feelings (e.g., ‘Think about a fire darting round a log in a fireplace’). The findings of dozens of such experimental studies indicate that rumination increases negative moods, particularly when people focus on negative aspects of their mood and their self (Watkins, 2008).

If rumination leads to sustained negative moods and even depression, why might people do it? According to one theory, it is evolutionarily adaptive to focus on negative events in order to solve

problems (Andrews & Thomson, 2009). Although the findings of many experimental studies suggest that rumination can interfere with solving puzzles presented in the laboratory, no tests have examined whether rumination facilitates solving more major and complex life stressors. In contrast to the idea that rumination is evolutionarily adaptive, a different model suggests that getting stuck on negative thoughts results from a basic inability to control the focus of thoughts. Research shows that people with depression do have a hard time ignoring irrelevant information to complete a task, particularly when they are asked to ignore negative information (Gotlib & Joormann, 2010). Although there is still some debate about why people ruminate, it is clear that people who often ruminate are more likely to become depressed.

## **Social and psychological factors in bipolar disorder**

Most people who experience a manic episode during their lifetime will also experience a major depressive episode, but not everyone will. For this reason, researchers often study the triggers of manic and depressive episodes separately within bipolar disorder.

### **Depression in bipolar disorder**

The triggers of depressive episodes in bipolar disorder appear to be similar to the triggers of major depressive episodes (Johnson, Cuellar, & Peckham, 2014). As in MDD, negative life events appear to be important in precipitating depressive episodes in bipolar disorder. Similarly, neuroticism, negative cognitive styles (Reilly-Harrington, Alloy, Fresco, & Whitehouse, 1999), expressed emotion (Miklowitz, Simoneau, Sachs-Ericsson, Warner, & Suddath, 1996) and lack of social support predict depressive symptoms in bipolar disorder.

### **Predictors of mania**

Two factors have been found to predict increases in manic symptoms over time: reward sensitivity and sleep deprivation. Both of these models integrate psychological and biological facets of vulnerability to mania.

#### **Reward sensitivity**

The first model suggests that mania reflects a disturbance in the reward system of the brain (Depue, Collins, & Luciano, 1996). Researchers have demonstrated that people with bipolar disorder describe themselves as highly responsive to rewards on a self-report measure (Meyer, Johnson, & Winters, 2001). Being highly reward sensitive has also been shown to predict the onset of bipolar disorder (Alloy et al., 2008) and a more severe course of mania after onset (Meyer et al., 2001). In addition, a particular kind of life event predicts increases in manic symptoms among people with bipolar I disorder — specifically, life events that involve attaining goals, such as gaining acceptance to university or getting married. How could successes like these promote increases in symptoms? Researchers have proposed that life events involving success may trigger cognitive changes in confidence, which then spiral into excessive goal pursuit (Johnson et al., 2012). This excessive goal pursuit may help trigger manic symptoms among people who are biologically vulnerable to bipolar disorder.

#### **Sleep deprivation**

Researchers using a range of approaches have documented the intricate relationship of mania to disruptions in sleep and circadian (daily) rhythms (Murray & Harvey, 2010). Experimental studies indicate that sleep deprivation can precede the onset of manic episodes. In one study, participants who were experiencing bipolar depression were asked to stay at a sleep centre, where they were kept awake all night. By the next morning, about 10 percent were experiencing at least mild symptoms of mania (Colombo, Benedetti, Barbini, Campori, & Smeraldi, 1999). In naturalistic studies, people often report that they had experienced a life event that disrupted their sleep just before the onset of manic episodes (Malkoff-Schwartz et al., 2000). Just as sleep deprivation can trigger manic symptoms, protecting sleep can help reduce symptoms of bipolar disorder (Frank, Swartz, & Kupfer, 2000). Sleep and circadian rhythm disruption appear to be important aspects of mania risk.

### **Integrating biological and social risk factors for depression: cytokines**

Depression rates are very high among people with medical conditions such as obesity, cardiovascular disorder, cancer, diabetes and Alzheimer's disease. For a long time, people thought that perhaps depression unfolded as a psychological response to the medical symptoms, but newer evidence suggests that this overlap in conditions may give us a clue about biological mechanisms in depression (Dantzer, O'Connor, Freund, Johnson, & Kelley, 2008).

The key to this model is that medical conditions often trigger elevations in cytokines. As described in chapter 1, cytokines are proteins that are released as part of an immune response. One set of these cytokines, pro-inflammatory cytokines, plays a vital role in wound healing and fighting off infection by triggering inflammation. In the short term, this inflammation is adaptive. Problems arise, though, when the inflammation becomes prolonged. The current theory is that people may vary in how well and quickly they recover from the influence of pro-inflammatory cytokines and that this prolonged response might be tied to depression.

Two of these pro-inflammatory cytokines, IL-1Beta and TNF-alpha, have been shown to cause a syndrome called sickness behaviour, which includes many of the symptoms that are seen in depression: decreased motor activity, reduced food consumption, social withdrawal, changes in sleep patterns and reduced interest in rewards (like sugar, in the case of rats). A wide range of studies support the idea that pro-inflammatory cytokines can cause sickness behaviour. For example, experimental administration of these two cytokines to animals triggers the symptoms of sickness behaviour, which can be relieved by administering antidepressant medications. In humans, drugs such as interferon, which are used for severe medical conditions such as cancer, also increase levels of pro-inflammatory cytokines. Between a third and a half of patients treated with interferon develop the symptoms of major depressive disorder (Raison, Capuron, & Miller, 2006). In naturalistic studies without experimental manipulation, many people with major depressive disorder and even those with no medical disorder, show elevated levels of cytokines (Raison et al., 2006). High levels of pro-inflammatory cytokines can predict changes in depressive mood over time (van den Biggelaar et al., 2007).

Although researchers do not think that all depressions are related to cytokines, cytokines may be important for understanding some of the triggers of depression. One theory is that pro-inflammatory cytokines might be the culprit among people who develop a first episode of depression in the context of a major medical illness (Dantzer et al., 2008). Others have argued that inflammation may play a role in how life events trigger symptoms (Slavich & Irwin, 2014). Major life stresses, and particularly interpersonal life stressors, can provoke increases in pro-inflammatory cytokines. In experimental studies, harsh social feedback has been shown to produce temporary increases in pro-inflammatory cytokines (Dickerson, Gruenewald, & Kemeny, 2009). The social stressors that commonly trigger depression may operate in part through this inflammatory mechanism (Slavich & Irwin, 2014). A growing body of research is focused on developing treatments to reduce the activity of pro-inflammatory cytokines (Dantzer et al., 2008).

#### **QUESTIONS**

1. Major illness is associated with psychological stresses that may also trigger depression. How have researchers been able to show evidence of a biological mechanism of pro-inflammatory cytokines?
2. How might hospitals use these findings when using interferon to treat individuals with cancer?

## **3.3 Treatment of mood disorders**

**LEARNING OUTCOME 3.3** Identify the medication and psychological treatments of mood disorders, as well as the current views of electroconvulsive therapy.

Most episodes of depression end after a few months, but the time may seem immeasurably longer to people with the disorder and to those close to them. With mania, even a few days of acute symptoms can create troubles for relationships and jobs. Moreover, suicide is a risk for people with mood disorders. Thus, it is important to treat mood disorders.

On one hand, approximately 1.7 million people (7.8 percent of the Australian population) filed at least one prescription for a PBS subsidised antidepressant (ABS, 2016b). On the other hand, 55.2 percent of individuals with an affective disorder received no mental health services.

## Psychological treatment of depression

Several different forms of psychological treatment have been shown to help relieve depression. These treatments are similar in being relatively brief (3–4 months of weekly sessions) and focused on the here and now. As with studies of aetiology, most of the research has focused on MDD. Available data, however, suggest that similar treatments can be helpful in the case of persistent depressive symptoms.

### Interpersonal psychotherapy

A treatment known as interpersonal psychotherapy (IPT) has fared well in clinical trials. As we described in chapter 1, IPT builds on the idea that depression is closely tied to interpersonal problems (Klerman, Weissman, Rounsaville, & Chevron, 1984). The core of the therapy is to examine major interpersonal problems, such as role transitions, interpersonal conflicts, bereavement and interpersonal isolation. Typically, the therapist and the patient focus on one or two such issues, with the goal of helping the person identify his or her feelings about these issues, make important decisions and effect changes to resolve problems related to these issues. Techniques include discussing interpersonal problems, exploring negative feelings and encouraging their expression, improving both verbal and nonverbal communications, problem solving and suggesting new and more satisfying modes of behaviour. Several studies have found that IPT is effective in relieving MDD (Elkin et al., 1989) and that it prevents relapse when continued after recovery (Frank et al., 1990).

### Cognitive-behavioural therapy

In keeping with their theory that depression is caused by negative schema and cognitive biases, Beck and associates developed cognitive-behavioural therapy (CBT) aimed at altering maladaptive thought patterns. To begin, the client is taught to understand how powerfully our thoughts can influence our moods and actions. To make this connection between their negative self-talk, mood and behaviour, the client might be asked to complete daily monitoring homework that involves recording their thoughts whenever they experience a negative mood. The therapist then tries to help the person with depression to change his or her opinions about the self, others and the world. When a person states that he or she is worthless because ‘nothing goes right and everything I try to do ends in a disaster’, the therapist helps the person look for evidence that contradicts this overgeneralisation, such as abilities that the person is overlooking or discounting. The therapist then teaches the person to challenge negative beliefs and to learn strategies that promote making realistic and positive assumptions. Often, the client is asked to practise challenging overly negative thoughts in his or her day-to-day life, recording an initially negative thought and then reconsidering whether this is the most accurate lens through which to view the situation (see table 3.6 for an example of a thought-monitoring homework assignment). Beck’s emphasis is on cognitive restructuring (i.e., persuading the person to think less negatively).

A review of meta-analyses of the efficacy of CBT showed a medium effect size supporting CBT for depression in adults (Cuijpers, Cristea, Karyotaki, Reijnders, & Huibers, 2016). The strategies that clients learn in CBT help diminish the risk of relapse even after therapy ends; this is an important issue given how common relapse is in MDD (Vittengl, Clark, Dunn, & Jarrett, 2007).

Computer-administered versions of CBT have been developed. Typically, these interventions include at least brief contact with a therapist to guide the initial assessment, to answer questions and to provide support and encouragement with the homework. Several randomised controlled trials provide evidence that computer-based CBT is effective compared to treatment as usual in which patients were instructed to seek help from other sources, as they normally would (Andrews, Cuijpers, Craske, McEvoy, & Titov, 2010). Because computer-based programs have varied in their effectiveness, it is important to ensure that consumers gain access to well-tested versions of computerised CBT (Spek et al., 2007).

**TABLE 3.6** An example of daily thought monitoring, a strategy commonly used in cognitive behaviour therapy

Date and time	Situation <i>What was happening?</i>	Negative emotion <i>Note type of emotion (e.g., sad, nervous, angry) and the intensity of the emotion (0–100)</i>	Automatic negative thought	How much did you believe this initial thought (0–100)?	Alternative thought <i>Is there another view of the situation?</i>	Re-rate your belief in the initial thought	Outcome <i>Note type of emotion felt and emotion intensity (0–100) after considering the alternative</i>
Tuesday morning	I made a mistake on a report at work.	Sad–90 Embarrassed–80	I always mess things up. I'm never going to be good at anything.	90	My boss didn't give me enough time to prepare the report. I could have done a better job with more time.	50	Relief–30 Sad–30
Wednesday dinner	Eating dinner at a restaurant. An old friend from high school was at the next table and didn't recognise me.	Sad–95	I'm a nobody.	100	I've changed my hair drastically since then. Many people don't recognise me, but maybe she would have been happy to see me if I had reminded her of who I was.	25	Sad–25
Thursday breakfast	My husband left for work without saying goodbye to me.	Sad–90	Even the people I love don't seem to notice me.	100	I know that he had a huge presentation and he gets stressed.	20	Sad–20

Behavioural activation (BA) is a technique used in CBT in which people are encouraged to engage in pleasant activities that might bolster positive thoughts about one's self and life. BA was originally developed as a standalone treatment and it is based on the idea that many of the risk factors for depression interfere with receiving positive reinforcement (Lewinsohn, 1974). That is, life events, low social support, marital distress, poverty and individual differences in social skills, personality and coping may all lead to low levels of positive reinforcement. As depression begins to unfold, inactivity, withdrawal and inertia are common symptoms and these symptoms diminish the already low levels of positive reinforcement even further. Consequently, the goal of BA is to increase participation in positively reinforcing activities so as to disrupt the spiral of depression, withdrawal and avoidance (Martell, Addis, & Jacobson, 2001).

BA has received a great deal of attention after positive findings in a study designed to identify the most effective ingredients in Beck's therapy (Jacobson & Gortner, 2000). The BA component of CBT has been found to perform as well as the full package does in relieving MDD and preventing relapse over a two-year follow-up period (Dobson et al., 2008). These findings challenge the notion that people must directly modify their negative thinking to alleviate depression and suggest instead that engaging in rewarding activities may be enough.

## CLINICAL CASE

### Challenging the negative thought in cognitive-behavioural therapy

The following dialogue is an example of one way that a therapist might begin to challenge a person's negative thoughts in CBT, although it would take several sessions to help a client learn the cognitive model and to identify overly negative thoughts.

**Therapist:** You said that you are a 'loser' because you and Roger got divorced. Now we already defined what it is to be a loser — not to achieve anything.

**Patient:** Right. That sounds really extreme.

**Therapist:** OK. Let's look at the evidence for and against the thought that you have achieved something. Draw a line down the centre of the page. On the top I'd like you to write, 'I have achieved some things.'

**Patient:** [draws line and writes statement]

**Therapist:** What is the evidence that you have achieved some things?

**Patient:** I graduated from university, I raised my son, I worked at the office, I have some friends and I exercise. I am reliable. I care about my friends.

**Therapist:** OK. Let's write all of that down. Now in the right column let's write down evidence against the thought that you have achieved some things.

**Patient:** Well, maybe it's irrational, but I would have to write down that I got divorced.

**Therapist:** OK. Now in looking at the evidence for and against your thought that you have achieved some things, how do you weigh it out? 50–50? Differently than 50–50?

**Patient:** I'd have to say it's 95% in favour of the positive thought.

**Therapist:** So, how much do you believe now that you have achieved some things?

**Patient:** 100%.

**Therapist:** And how much do you believe that you are a failure because you got divorced?

**Patient:** Maybe I'm not a failure, but the marriage failed. I'd give myself about 10%.

*Note:* As is typical, this dialogue challenges some, but not all, negative thoughts. Future sessions are likely to examine other negative thoughts.

*Source:* R. Leahy, *Cognitive Therapy Techniques: A Practitioner's Guide* [New York: Guilford Press, 2003], Copyright Guilford Press. Reprinted with permission of the Guilford Press.

### QUESTIONS

1. Imagine you are the therapist in this clinical case. How could you explain the cognitive model of depression and the aims of CBT to this patient?
2. What two CBT-based take-home tasks could the therapist set for this patient?

## Behavioural couples therapy

Because depression is often tied to relationship problems, including marital distress, researchers have studied **behavioural couples therapy** as a treatment for depression. In this approach, researchers work with both members of a couple to improve communication and relationship satisfaction. Findings indicate that when a person with depression is also experiencing marital distress, behavioural couples therapy is as effective in relieving depression as individual therapy (Jacobson, Dobson, Fruzzetti, & Schmalings, 1991) or antidepressant medication (Barbato & D'Avanzo, 2008). As you might expect, marital therapy has the advantage of relieving relationship distress more than does individual therapy.

## Third wave cognitive therapies

So-called first wave cognitive therapy approaches were largely focused on behavioural techniques such as BA, and the current form of CBT with its focus on changing thoughts is known as the second wave. Third wave cognitive therapies such as **mindfulness-based cognitive therapy (MBCT)**, acceptance and commitment therapy (ACT) and dialectical behaviour therapy (DBT) are increasing in popularity

as treatments for depression. Similar to Beck's cognitive model of depression, third wave therapies also recognise that belief in the content of negative thoughts contributes to low mood and self-defeating patterns of behaviour. Here, individuals are 'fused' with their thoughts if they hold them as literal truths and allow them to dictate their behaviour. However, this wave of therapies suggests that changing patients' relationships to their thoughts, rather than challenging the content of thoughts, can improve depression.

The goal of third wave therapies is to assist patients to adopt a 'decentred' or 'defused' perspective by teaching them to view thoughts as 'mental events' rather than accurate reflections of reality or the self. The client is helped to accept that the experience of negative thoughts and feelings is a normal part of human existence and in doing so reduce feelings of distress that come with the struggle to be rid of painful experiences. It is believed that by creating a detached relationship to thoughts through mindfulness meditation and defusion techniques (e.g., saying to oneself 'thoughts are not facts'), patients will be less likely to experience depression and spend more time engaging in positive, value-driven activities.

Despite the increasing popularity of third wave approaches, there is as yet little high-quality evidence to support their efficacy as treatments for depression. A Cochrane review found some evidence that third wave approaches are more effective than treatment as usual for MDD, but it only included four small studies that differed in patient group, interventions and control conditions used (Churchill et al., 2013). The third wave approach with the most empirical support is MBCT: a meta-analysis of 11 studies showed it is helpful at preventing depressive relapses, particularly in individuals who have three or more previous episodes of MDD (Galante, Iribarren, & Pearce, 2013). Given that third wave therapies have only recently seen an increase in popularity, it may be some time before enough high-quality evidence is available by which to judge its effectiveness.

### **Mindfulness in your daily routine**

Mindfulness is a type of activity that teaches people to have an intentional, non-judgemental awareness of the present moment. By learning to live in the here and now, individuals may be less vulnerable to rumination and negative self-talk. The following exercise, adapted from Harris (2009), shows how people can practise mindfulness in their day-to-day lives:

Choose an activity that you do on a daily basis, such as brushing your teeth, showering, eating cereal, or making the bed. The next time you do this activity, totally focus your attention on what you can experience through your five senses: taste, touch, smell, sight, sound. Try to notice what you are experiencing in every step of the process with openness and curiosity.

Say you wash the dishes every night after dinner. Next time, try to pay attention to the sound of the water splashing in the sink and the clinking sounds as you stack clean glasses. Notice the smell of detergent and the feel of the warm water. Watch closely the patterns the bubbles make on the surface and the ripples that spread out as you dunk a new item. Feel the movement of your arms as you scrub.

If you find yourself getting distracted by your thoughts about your day (and it happens every day — that's just what minds do), gently acknowledge your thoughts, let it go and bring your attention back to your dishes.

(Adapted from Harris, 2009)

Try this exercise yourself and see what you notice about paying attention to your daily routines in this way.

## **Psychological treatment of bipolar disorder**

Medication is a necessary part of treatment for bipolar disorder, but psychological treatments can supplement medication to help address many of its associated social and psychological problems. These psychotherapies can also help reduce depressive symptoms in bipolar disorder.

Educating people about their illness is a common component of treating many disorders, including bipolar disorder and schizophrenia. **Psychoeducational approaches** typically help people learn about the symptoms of the disorder, the expected time course of symptoms, the biological and psychological triggers for symptoms and treatment strategies. Studies confirm that education about bipolar disorder can help people adhere to treatment with medications such as lithium (Colom et al., 2003). This is an important goal because as many as half of people being treated for bipolar disorder do not take medication consistently (Regier, Narrow, Rae, & Manderscheid, 1993). Psychoeducation can help clients

understand the rationale for taking these medications even after symptoms dissipate, can foster hope that the medications will help and can target the internalised stigma that many clients experience when taking a medication such as lithium. Beyond helping people be more adherent with their medications, psychoeducational programs have been shown to help people avoid hospitalisation (Morris et al., 2007).

Several other types of therapy are designed to help build skills and reduce symptoms for those with bipolar disorder. Both CBT and family-focused therapy (FFT) have received particularly strong support (Lam et al., 2000). CBT draws on many of the types of techniques that are used in major depressive disorder, with some additional content designed to address the early signs of manic episodes. FFT aims to educate the family about the illness, enhance family communication and develop problem-solving skills (Miklowitz & Goldstein, 1997). **Interpersonal and social rhythm therapy (IPSRT)** was designed to stabilise daily rhythms and thus avoid disruptions to the circadian system which can trigger episodes of mania (Frank, Swartz, & Kupfer, 2000).

In one large study, researchers recruited people who had bipolar disorder and who were depressed at treatment entry (Miklowitz et al., 2007). All of the patients in the trial received intensive medication treatment because researchers were interested in whether adding psychotherapy to medication treatment for bipolar disorder would be helpful. Patients were randomly assigned to receive either psychotherapy or three sessions of a control treatment called collaborative care (psychoeducation about bipolar disorder). The 163 patients in the psychotherapy condition were further assigned to receive CBT, FFT or IPSRT. Each type of psychotherapy helped relieve depression more than the collaborative care condition did. There was no evidence that CBT, FFT or IPSRT differed in their effects on depression. These findings suggest that several types of psychotherapy are helpful for bipolar depression.

Even with best practice biological and psychological treatment for bipolar disorder, depressive and manic relapses are common (Miklowitz, 2008). It has been suggested that given the chronic nature of bipolar disorder, psychological treatments should focus on means to improve quality of life as well as manage symptoms (Murray & Michalak, 2012). Recovery-oriented psychoeducation (focused on living well despite symptoms) and third wave therapies (focused on accepting painful experiences beyond one's control) are the focus of novel investigations to improve quality of life in bipolar disorder (e.g., [www.bdwellness.com](http://www.bdwellness.com); Murray et al., 2015; Todd, Jones, Hart, & Lobban, 2014).

## Biological treatment of mood disorders

A variety of biological therapies are used to treat depression and mania. The two major biological treatments are electroconvulsive therapy and drugs. We will also briefly discuss transcranial magnetic stimulation, a technique that is approved by the Therapeutic Goods Administration (TGA) for a small subset of people with MDD.

### Electroconvulsive therapy for depression

Perhaps the most controversial treatment for MDD is electroconvulsive therapy (ECT), due in part to its depiction in films such as *One Flew Over the Cuckoo's Nest*. For the most part now, ECT is only used to treat MDD that has not responded to medication. ECT entails deliberately inducing a momentary seizure by passing a 70- to 130-volt current through the patient's brain. Formerly, electrodes were placed on each side of the forehead, a method known as bilateral ECT. Today, *unilateral ECT*, in which the current passes only through the non-dominant (typically the right) cerebral hemisphere, is often used because side effects are less pronounced than with bilateral ECT (McCall, Reboussin, Weiner, & Sackeim, 2000). In the past, the patient was usually awake until the current triggered the seizure and the electric shock often created frightening contortions of the body, sometimes even causing bone fractures. Now the patient is given a muscle relaxant before the current is applied so that they sleep through the procedure and the convulsive spasms of muscles are barely perceptible. The patient awakens a few minutes later remembering nothing about the treatment. Typically, patients receive between 6 and 12 treatments, spaced several days apart.

Even with these improvements in procedures, inducing a seizure is drastic treatment. Why should anyone agree to undergo such radical therapy? The answer is simple. ECT is more powerful than antidepressant medications for the treatment of depression (UK ECT Review Group, 2003), particularly

when psychotic features are present (Sackeim & Lisanby, 2001). Most professionals acknowledge that people undergoing ECT face some risks of short-term confusion and memory loss. It is fairly common for patients to have no memory of the period during which they received ECT and sometimes for the weeks surrounding the procedure. Unilateral ECT produces fewer cognitive side effects than bilateral ECT does. Nonetheless, even unilateral ECT is associated with deficits in cognitive functioning 6 months after treatment (Sackeim et al., 2007). In any case, clinicians typically resort to ECT only if less drastic treatments have failed. Given that suicide is a real possibility among people who are depressed, many experts regard the use of ECT after other treatments have failed as a responsible approach.

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Electroconvulsive therapy is an effective treatment for depression that has not responded to medication. Using unilateral shock and muscle relaxants has reduced undesirable side effects.



### Medications for depressive disorders

Drugs are the most commonly used and best-researched treatments — biological or otherwise — for depressive disorders (and, as we will see, for bipolar disorders as well). Antidepressant medications are recommended for moderately severe depression in Australian and New Zealand clinical guidelines (Malhi et al., 2015). As shown in table 3.7, there are four major categories of **antidepressant** drugs: **monoamine oxidase inhibitors (MAOIs)**, **tricyclic antidepressants**, **selective serotonin reuptake inhibitors (SSRIs)** and **serotonin noradrenaline reuptake inhibitors (SNRIs)** (see figure 3.5 for more detail on tricyclic antidepressants and SSRIs). The superiority of one antidepressant over another has not been established due to heterogeneity of the populations studied. Dozens of double-blind studies have shown these medications to be effective in treating depressive disorders, with 50–70 percent of people who complete treatment showing major improvement (Nemeroff & Schatzberg, 1998).

**TABLE 3.7 Medications for treating mood disorders**

Use	Category	Generic name	Brand name
Antidepressants	MAO inhibitors	Tranylcypromine	Parnate
	Tricyclic antidepressants	Imipramine Amitriptyline	Tofranil Endep
	Selective serotonin reuptake inhibitor (SSRI)	Fluoxetine Sertraline	Prozac, Lovan Zoloft
	Serotonin noradrenaline reuptake inhibitor (SNRI)	Venlafaxine Desvenlafaxine Duloxetine	Effexor Pristiq Cymbalta
Mood stabilisers		Lithium	Lithicarb, Quilonum SR
	Anticonvulsants	Sodium valproate Carbamazepine Lamotrigine	Epilim, Valpro Tegretol, Teril Lamictal
	Antipsychotics	Olanzapine Quetiapine Aripiprazole Risperidone	Zyprexa Seroquel Abilify Risperdal

Treatment guidelines recommend continuing antidepressant medications for at least 12 months after a depressive episode ends — and longer if a person has experienced several episodes. Continuing antidepressants after remission lowers the risk of recurrence from approximately 40 percent to about 20 percent (Geddes et al., 2003). To prevent recurrence, medication doses should be as high as those offered during acute treatment.

In the past several years, a number of controversies have developed regarding antidepressants. One major concern is that antidepressants do not appear to be more effective than placebos for relieving mild or moderate symptoms of major depressive disorder. Antidepressants do offer a clear advantage compared to placebos in the treatment of severe major depressive disorder (Fournier et al., 2010) or persistent depressive disorder (Keller et al., 2000).

Beyond the evidence that these medications may not be helpful for those with mild levels of depression, published studies may overestimate how many people respond well to antidepressant medications. When pharmaceutical companies conduct studies to apply for either initial approval to market a medication or to support a change in the use of a medication in the United States, the data must be filed with the Food and Drug Administration (FDA). One research team examined what happened to the data from antidepressant studies conducted between 1987 and 2004 (Turner, Matthews, Linardatos, Tell, & Rosenthal, 2008). Of the 74 studies conducted, 51 percent were rated as having positive findings (i.e., supported the use of the antidepressant). All but one of the studies with positive findings were published. Of the studies with negative findings, less than half were published, and even in those reports, the published reports described the findings as positive even though the FDA had rated them as neutral or negative. Overall, then, the published findings were much more positive than the data in the FDA files. Similar criticisms have been levied towards the TGA in Australia.

In the face of these discouraging reports, one strategy is to conduct studies focused on what to do when a person does not gain relief from a first round of an antidepressant. In an attempt to answer this type of question, the STAR-D (Sequenced Treatment Alternatives to Relieve Depression) trial examined antidepressant response among 3671 patients across 41 sites, including 18 primary care facilities (Rush et al., 2006). In sharp contrast to the types of clean, non-comorbid depression histories reported in most medication trials, most of the patients enrolled in STAR-D suffered from chronic or recurrent depression, had comorbid psychiatric conditions and had already received some (unsuccessful) treatment for the current episode. Rather than assessing whether antidepressant medication or psychotherapy was more helpful than a placebo

treatment, the goal of the study was to consider the types of practical questions that physicians face in daily practice. Patients were all started on citalopram, an SSRI. If they did not respond to citalopram, they were offered (1) a choice of a different medication to replace the citalopram, (2) a chance to add a second medication to the citalopram or (3) CBT if they were willing to pay part of the cost. The researchers provided patients who did not respond with a third type of antidepressant and if needed, even a fourth.

The findings were sobering. Only about one-third of patients achieved full symptom relief when treated with citalopram (Trivedi et al., 2006). Among those who did not respond to citalopram, very few wanted to pay for CBT and only 30.6 percent who received a second medication (either alone or as a supplement to citalopram) achieved remission. Across the course of treatments, relapse rates were high, so that even with the complex array of treatments offered, only 43 percent of people achieved sustained recovery (Nelson, 2006). The findings from this study highlight the need to develop new treatments for those who do not respond well to currently available treatments.

Among patients who are prescribed an antidepressant, 28 percent stop taking the medication within the first month (Lin et al., 1995), most commonly because they have a hard time tolerating the side effects such as dizziness, headaches, erectile dysfunction or gastrointestinal complaints (Thase & Rush, 1997). The MAOIs are the least used antidepressants because of their potentially life-threatening side effects if combined with certain foods or beverages. The SSRIs have become the most commonly prescribed antidepressants because they tend to produce fewer side effects than the other classes of antidepressants (Enserink, 1999). Nonetheless, there is specific concern about SSRI use and the potential for suicidality in children, adolescents and young adults; researchers continue to look at this important issue, as findings have been controversial (see the chapter on disorders of childhood). The impact of side effects on medication adherence in depression is of particular concern given antidepressants can take four weeks to show full effect, whereas side effects are typically felt in the first week of use.

### **Transcranial magnetic stimulation for depression**

In 2007, the TGA approved **transcranial magnetic stimulation (rTMS)** for a small subset of those with depression: patients who have failed to respond to at least two adequate trials of an antidepressant medication. In this approach, an electromagnetic coil is placed against the scalp and intermittent pulses of magnetic energy are used to increase activity in the dorsolateral prefrontal cortex. Typical treatment lasts 30 minutes, with daily doses delivered for 5 to 10 days. In most randomised controlled trials, researchers have compared the treatment to a false treatment in which the device is placed against the scalp at enough of an angle that the magnetic pulse will not increase brain activity. Multiple randomised controlled trials suggest that rTMS can help relieve treatment-resistant depression compared to the false treatment (Slotema, Blom, Hoek, & Sommer, 2010).

## **Comparing treatments for major depressive disorder**

Combining psychotherapy and antidepressant medications bolsters the odds of recovery by more than 10–20 percent above either psychotherapy or medications alone for most people with depression (Hollon, Thase, & Markowitz, 2002). One study found that even offering cognitive therapy (a form of CBT focused specifically on cognitive components) by telephone for those beginning antidepressants could improve outcomes compared to medication alone (Simon, 2009). Each treatment offers unique advantages. Antidepressants work more quickly than psychotherapy, thus providing immediate relief. Psychotherapy may take longer but may help people learn skills that they can use after treatment is finished to protect against recurrent depressive episodes.

Many patients are interested in knowing whether medications or therapy will be more effective in relieving symptoms. In the most careful study to date comparing cognitive therapy to antidepressants, researchers focused on the treatment of severe depression. Two hundred and forty patients with severe depression were randomly assigned to receive antidepressant medication, cognitive therapy or a placebo for four months. Those who recovered were followed for another 12 months. Cognitive therapy (CT) was as effective as antidepressant medication for severe depression and both treatments were more effective than placebo.

CT had two advantages: it was less expensive than medication and over the long term it helped protect against relapse once treatment was finished (Hollon et al., 2005).

## Medications for bipolar disorder

Medications that reduce manic symptoms are called *mood-stabilising medications*. The mood-stabilising properties of **lithium**, a naturally occurring chemical element, were identified in 1949 by John Cade, an Australian doctor.

Up to 80 percent of people with bipolar I disorder experience at least mild benefit from taking this drug (Prien & Potter, 1993). Even though symptoms are usually decreased with medications, most patients continue to experience at least mild manic and depressive symptoms. Results of a major meta-analysis indicated that 40 percent of people relapsed while taking lithium as compared to 60 percent while taking a placebo (Geddes, Burgess, Hawton, Jamison, & Goodwin, 2004). Because of possibly serious side effects, lithium has to be prescribed and used very carefully. Lithium levels that are too high can be toxic, so patients taking lithium must have regular blood tests.

Two classes of medications other than lithium are used for the treatment of acute mania: anticonvulsant (antiseizure) medications such as sodium valproate (Epilim) and antipsychotic medications such as olanzapine (Zyprexa). These other treatments are recommended for people who are unable to tolerate lithium's side effects. Like lithium, these medications help reduce mania and, to some extent, depression. Unfortunately, even these medications have serious side effects. As one example, anticonvulsants have been found to be related to a small increase in suicidal ideation compared to rates for placebo (Food and Drug Administration, 2008).

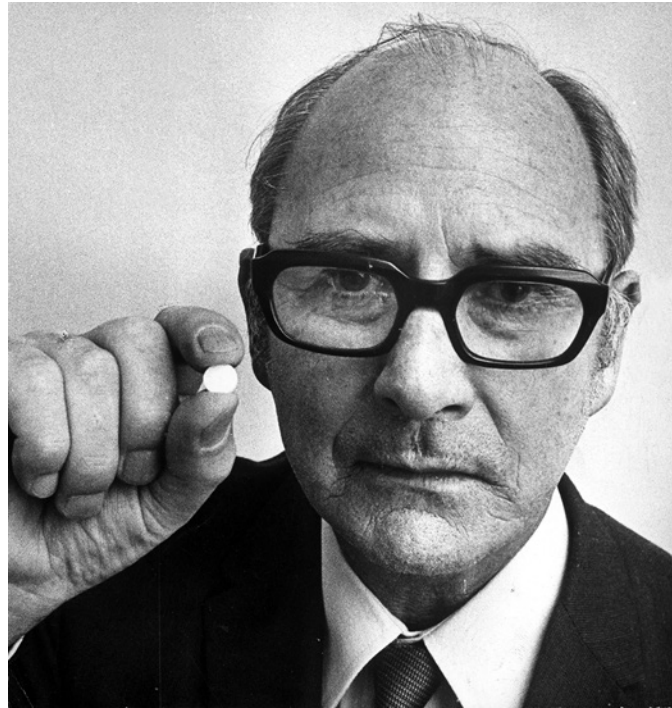
Typically, lithium is used in combination with other medications. Because lithium takes effect gradually, therapy for acute mania often begins with both lithium and an antipsychotic medication, such as olanzapine, which has an immediate calming effect (Scherk, Pajonk, & Leucht, 2007).

The mood-stabilising medications used to treat mania also help relieve depression. Nonetheless, many people continue to experience depression even when taking a mood-stabilising medication such as lithium. For these people, an antidepressant medication is often added to the regimen (Sachs & Thase, 2000), but two potential issues are associated with this practice. First, it is not clear whether antidepressants actually help reduce depression among persons who are already taking a mood stabiliser (the first type of treatment provided in bipolar disorder). Second, among people with bipolar disorder, antidepressants are related to a modest increase in the risk of a manic episode if taken without a mood stabiliser (Pacchiarotti et al., 2013).

Clinical guidelines recommend that individuals with bipolar disorder continue to take mood stabilising medications for the duration of their life in order to prevent future mood episodes or reduce their impact (Malhi et al., 2015).

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Australian Dr John Cade identified the mood-stabilising properties of lithium in 1949.



## RETURNING TO OPENING SCENARIO

### Treatment decisions for Mary

Mary, the woman described at the beginning of this chapter, reported increasing problems because of her depression. Accordingly, her therapist referred her to a psychiatrist, who prescribed fluoxetine (Prozac). Both the psychologist and the psychiatrist agreed that medication might help by quickly relieving her symptoms. But after two weeks, Mary decided she did not want to continue taking Prozac because she found the side effects uncomfortable and did not like the idea of taking medication over the long term. She had not gotten much relief, maybe because her concerns about medication had led her to skip many doses.

With so many different types of treatment available, determining the best therapy for a given client can be a challenge. Mary had experienced a major life event and transition, suggesting that interpersonal psychotherapy might fit. Mary was blaming herself for her job loss and other issues and no longer engaging in activities she enjoyed, suggesting that CBT might help. Third wave cognitive therapies such as MBCT may help Mary decentre from unhelpful self-talk, reducing her distress over negative thoughts. Marital conflicts suggested behavioural couples therapy could be appropriate.

How does a therapist choose which approach to use? Sometimes this decision reflects the personal preferences and training of the therapist. Ideally, the approach incorporates the treatment preferences of the client as well. Her therapist began CBT, in the belief that Mary's tendency to blame herself excessively when things went wrong was contributing to her depression. CBT helped her learn to identify and challenge irrationally negative cognitions about herself. Therapy began by helping her identify times in her day-to-day life when her sad moods could be explained by overly negative conclusions about small events. For example, when her children would misbehave, Mary would quickly assume this was evidence that she was a bad mother. The therapist also encouraged Mary to schedule time for activities she used to enjoy, such as socialising with friends. Over time, Mary began to examine and challenge long-held beliefs about her lack of competence. Through spending more time with friends, Mary received positive reinforcement about her worth and withdrew less. By the end of 16 weeks of treatment, she had obtained relief from her depression.

### QUESTIONS

1. What information about the benefits and side effects of antidepressants would Mary's GP need to give her in order to help her make an informed decision?
2. Imagine you are the therapist in this case. Which psychological treatment for depression would you choose to use with Mary and why?

## A final note on treatment

Many researchers are studying how treatments influence the psychological and biological mechanisms that seem to drive depression. For example, researchers have shown that antidepressants reduce negative thinking among persons with depression well before they influence mood state (Harmer, Goodwin, & Cowen, 2009). Researchers also have shown that successful treatments, whether with psychotherapy, medications or ECT, change activity in the brain regions related to depression (Goldapple et al., 2004; Nobler et al., 2001).

In addition to considering the mechanisms through which treatments work, researchers have been developing new approaches to treatment. One newer approach is **deep brain stimulation**, a technique that involves implanting electrodes into the brain (Mayberg et al., 2005). By applying a small current to the electrodes, activity within that brain region can be manipulated. These studies are typically conducted only with small numbers of people who did not respond to other treatments for depression and it is too early to consider deep brain stimulation as a standard treatment approach. Nonetheless, deep brain stimulation studies provide an important test of whether changing activity in a given region can help relieve depressive symptoms. Some researchers found that manipulating activity in the anterior cingulate and the nucleus accumbens using deep brain stimulation led to decreased depressive symptoms in about half of individuals who had failed to respond to previous depression treatments

(Bewernick et al., 2010; Holtzheimer & Mayberg, 2011). The findings support the idea that the anterior cingulate and the nucleus accumbens play an important role in depression. Understanding more about how psychological and medication treatments change underlying neurobiological processes may help us refine treatments for the future.

#### CLINICAL CASE

##### Steven

“Shannon Neal can instantly tell you the best night of her life: Tuesday, December 23, 2003, the Hinsdale Academy debutante ball. Her father, Steven Neal, a 54-year-old political columnist for The Chicago Sun-Times, was in his tux, white gloves, and tie. ‘My dad ‘walked me down and took a little bow,’ she said, and then the two of them goofed it up on the dance floor as they laughed and laughed. A few weeks later, Mr. Neal parked his car in his garage, turned on the motor and waited until carbon monoxide filled the enclosed space and took his breath, and his life, away.”

He had been under stress as he finished a book and had been hospitalized for heart problems. “Still, those who knew him were blindsided. ‘If I had just 30 seconds with him now,’ Ms. Neal said of her father, ‘I would want all these answers.’”

*Source:* Cohen (2008).

#### QUESTIONS

1. What risk factors for suicide are present in this case?
2. What are some of the risks faced by Ms Neal, as a person bereaved by suicide?

## 3.4 Suicide

**LEARNING OUTCOME 3.4** Explain the epidemiology and risk factors associated with suicide, as well as methods for preventing suicide.

No other kind of death leaves friends and relatives with such long-lasting feelings of distress, shame, guilt and puzzlement as does suicide (Gallo & Pfeffer, 2003). Survivors have an especially high mortality rate in the year after the suicide of a loved one.

We will focus on quantitative research on suicide, but those who study suicide learn from many different sources. Many philosophers have written searchingly on the topic, including Descartes, Voltaire, Kant, Heidegger and Camus. In addition, novelists such as Herman Melville and Leo Tolstoy have provided insights on suicide, as have writers who have died by suicide, such as Virginia Woolf and Sylvia Plath. The study of suicide involves many different ethical questions and forces people to consider their own views on life and death.

We begin by defining terms (see table 3.8). Suicidal ideation refers to thoughts of killing oneself and is much more common than attempted or completed suicide. Suicide attempts involve behaviours that are intended to cause death. Most

Writers who died by suicide, such as Sylvia Plath, have provided insights into the causes of suicide. In perhaps an act that is at least partially related to the heritability of suicide, Sylvia Plath’s son also died by suicide.



suicide attempts do not result in death. **Suicide** involves behaviours that are intended to cause death and actually do so. **Non-suicidal self-injury** involves behaviours that are meant to cause immediate bodily harm but are not intended to cause death (see focus on discovery 3.6).

**TABLE 3.8** Key terms in the study of suicidality

**Suicidal ideation:** thoughts of killing oneself

**Suicide attempt:** behaviour intended to kill oneself

**Suicide:** death from deliberate self-injury

**Non-suicidal self-injury:** behaviours intended to injure oneself without intent to die

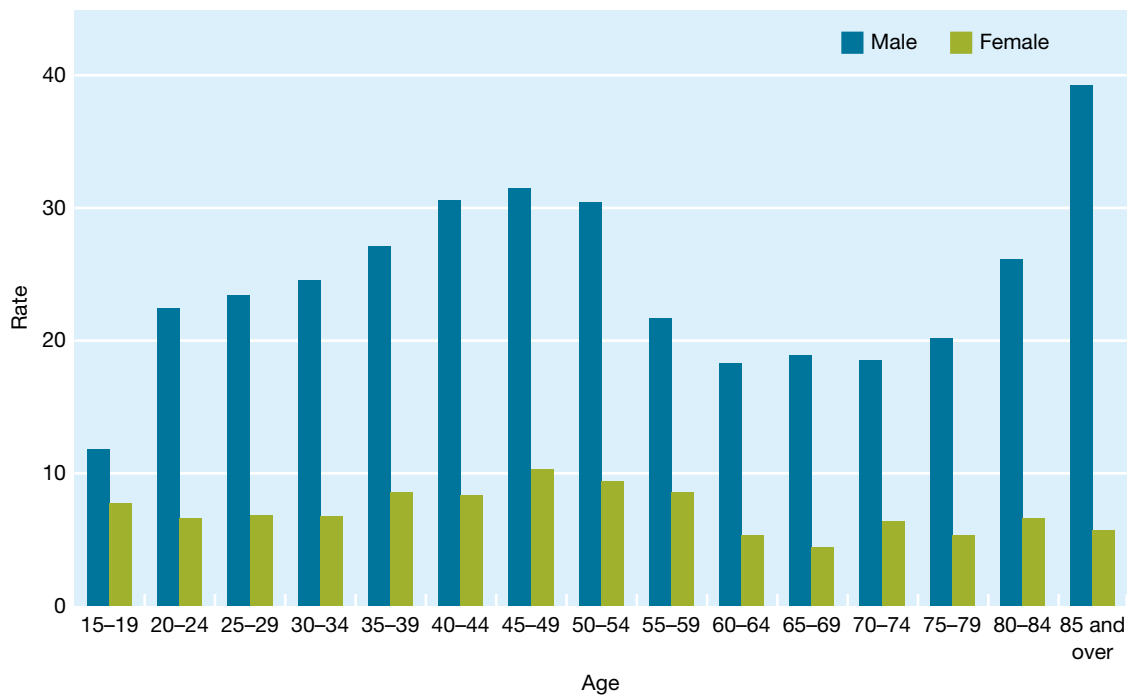
## Epidemiology of suicide and suicide attempts

Suicide rates may be grossly underestimated because the circumstances of some deaths are ambiguous; for example, a seemingly accidental death may have involved suicidal intentions. Nonetheless, it has been estimated that suicide is the thirteenth leading cause of death in Australia (ABS, 2016a).

Studies on the epidemiology of suicidality suggest the following.

- In Australia, the overall suicide rate is about 12.6 per 100 000 in a given year (ABS, 2016a).
- Worldwide, about 9 percent of people report suicidal ideation at least once in their lives and 2.5 percent have made at least one suicide attempt.
- Suicide rates are higher in regions where more people own guns. Guns are by far the most common means of suicide in the United States (Arias, Anderson, Kung, Murphy, & Kochanek, 2003), accounting for about 60 percent of all suicides. States and countries where more people own guns tend to have higher suicide rates (Miller & Hemenway, 1999).
- Men are three times more likely than women to die by suicide (ABS, 2016a).
- Women are more likely than men are to make suicide attempts that do not result in death (Nock & Mendes, 2008).
- Men usually attempt suicide using guns or hanging; women are more likely to take an overdose of drugs, a less lethal method, which may account for their lower rate of completed suicide.
- The suicide rate increases in old age. The highest rates of suicide in Australia are for males over age 85, with the next highest rate being for males aged 45 to 49 years (ABS, 2016a).
- The rates of suicide for adolescents and children in Australia are increasing dramatically but are still far below the rates of adults (see figure 3.10). Some estimates suggest that at least 40 percent of children and adolescents experience suicidal ideation at least once. Because young people are less likely to die from other causes, suicide ranks as the leading cause of death among those aged 5 to 17 (ABS, 2016a).
- Being divorced or widowed elevates suicide risk four or fivefold.
- The rate of death by suicide for Aboriginal and Torres Strait Islander peoples is almost double the rate of non-Indigenous Australians (25.5 deaths per 100 000 persons). Suicide is the leading cause of death for Indigenous Australians aged between 15 and 34 years of age (ABS, 2016a).

**FIGURE 3.10** Annual deaths due to suicides per 100 000 people



### FOCUS ON DISCOVERY 3.6

#### Non-suicidal self-injury

Non-suicidal self-injury (NSSI) is more common than previously thought (Nock, 2010) and contrary to previous thinking, it is practised by many who do not have borderline personality disorder (Nock, Kazdin, Hiripi, & Kessler, 2006). Here we define this behaviour and give some reasons it occurs.

There are two key issues to consider in defining NSSI. The first is that the person did not intend to cause death. The second is that the behaviour is designed to immediately cause injury. Most commonly, people cut, hit or burn their body (Franklin et al., 2010). When surveyed, anywhere from 13 to 45 percent of adolescents report having engaged in NSSI, but the higher estimates may be due to overly broad definitions. For example, some studies will include 'scratching to the point of bleeding', without ruling out insect bites or other reasons people might be scratching. Without a doubt, though, there is a group of people who engage in serious attempts to hurt themselves. The modal profile is of a person who tries NSSI infrequently (less than 10 times) during early adolescence and then stops (Nock, 2009). A subset of people seem to persist in self-injury, sometimes reporting more than 50 incidents of self-injury per year, and it is this profile of persistent NSSI that researchers are working to understand (Nock & Prinstein, 2004).

Actress and singer Demi Lovato, who has struggled with bipolar disorder, spoke of engaging in non-suicidal self-injury as a response to intense emotional pain.



In a comprehensive review, Nock (2010) argues that people will hurt themselves for many different reasons. For some, the injury seems to help quell negative emotions, such as anger. Some report feeling satisfied after self-injury because they have given themselves punishment that they believe they deserved. The behaviour can also be reinforcing interpersonally — others may respond by increasing support or by reducing aggression. Several studies provide evidence that those who are prone to self-injury do seem to experience more intense emotions and provide more intense psychophysiological responses to stress than others do; believe that they deserve to be punished; and report difficulty managing relationships constructively (Nock & Mendes, 2008). In one study, people who engaged in NSSI were asked to record feelings, events and NSSI incidents daily over time. Feelings of self-hatred and of being rejected were common just before incidents of self-injury took place (Nock, Prinstein, & Sterba, 2009). Taken together, NSSI may be reinforcing both psychologically (by relieving feelings of self-hatred and anger) and socially (by eliciting more supportive reactions from others). It is likely to require integrated social, emotional and cognitive research to fully understand this complex behavioural pattern. To foster more attention to this issue, the DSM-5 includes an NSSI diagnosis in the appendix as a condition for further study.

### QUESTIONS

1. How is the profile of an individual at risk of NSSI different to that of an individual at risk of suicide?
2. Based on the reasons for engaging in NSSI identified in the literature, what hypotheses could you come up with for possible intervention strategies?

## Risk factors for suicide

Suicide is such a complex and multifaceted act that no single model can hope to explain it. Myths about suicide abound, highlighting the need for careful research (see table 3.9). Increasingly, those who have survived suicide attempts have joined public advocacy and support movements to help inform this field and their perspectives may enhance our ability to understand factors that lead up to suicide attempts (see [livethroughthis.org](http://livethroughthis.org)).

**TABLE 3.9** Myths about suicide

Common myth	Contrary evidence
People who discuss suicide will not actually commit suicide.	Up to three-quarters of those who take their own lives communicate their intention beforehand.
Suicide is committed without warning.	People usually give many warnings, such as saying that the world would be better off without them or making unexpected and inexplicable gifts of highly valued possessions.
Suicidal people want to die.	Most people are thankful after suicide is prevented.
People who attempt suicide by low lethality means are not serious about killing themselves.	Many people are not well informed about pill dosages or human anatomy. Because of this misinformation, people who really want to die sometimes make non-lethal attempts.

*Source:* Fremouw, De Perczel, & Ellis (1990).

## Psychological disorders

Suicide is discussed in this chapter because many persons with mood disorders have suicidal thoughts and some engage in suicidal behaviours. More than half of those who attempt suicide are depressed at the time of the act (Centers for Disease Control and Prevention, 2006) and as many as 15 percent of people who have been hospitalised with depression ultimately die from suicide (Angst et al., 2002). Other psychological disorders also are important in understanding suicide: as many as 90 percent of people who attempt suicide are suffering from a psychological disorder. About 5–7 percent of people diagnosed with bipolar disorder and 5 percent diagnosed with schizophrenia eventually die from suicide (Palmer, Pankratz, & Bostwick, 2005; Tondo, Isacson, & Baldessarini, 2003). Impulse control disorders, substance

use disorders, PTSD and borderline personality disorder are also each related to a higher risk of suicidal actions (Linehan, 1997; Nock & Mendes, 2008; Nock et al., 2009). Even less severe psychological disorders, such as panic disorder and eating disorders, are associated with elevated risk of suicide (Linehan, 1997; Schmidt, Woolaway-Bickel, & Bates, 2000). With most of these disorders, though, suicides are most likely when a person is experiencing comorbid depression (Angst et al., 2002; Schmidt et al., 2000). Although understanding suicide within the context of psychological disorders is extremely important, most people with psychological disorders do not die from suicide.

### Neurobiological factors

Twin studies suggest that heritability is about 48 percent for suicide attempts (Joiner, Brown, & Wingate, 2005). That is, genetic factors may explain about half of the variance in who will attempt suicide. Adoption studies also support the heritability of suicidality.

Just as they are with depression, serotonin and cortisol are relevant to understanding suicidality. Researchers have found links between serotonin and suicide using an array of paradigms (Mann et al., 2000). Serotonin dysfunction appears to be particularly relevant for understanding violent suicide (Roy, 1994; Winchel, Stanley, & Stanley, 1990). Beyond the serotonin system, among patients with MDD, those who had an abnormal dexamethasone suppression test response had a 14-fold increase in the risk of suicide over the next 14 years (Coryell & Schlesser, 2001). Cortisol dysregulation and serotonin deficits, then, appear to be important predictors of suicidality.

### Social factors

Economic and social events have been shown to influence suicide rates. As one example, across the past 100 years, suicide rates have been shown to increase modestly during economic recessions (Luo, Florence, Quispe-Agnoli, Ouyang, & Crosby, 2011). Some of the strongest evidence for the role of the social environment in suicide comes from the major effects of media reports of suicide. In one example of these effects, suicides rose 12 percent in the month after Marilyn Monroe's suicide (Phillips, 1985). A review of 293 studies found that media coverage of a celebrity suicide is much more likely to spark an increase in suicidality than coverage of a non-celebrity suicide (Stack, 2000). Media reports of natural deaths of famous people are not followed by increases in suicide, suggesting that it is not grief per se that is the influential factor (Phillips, 1974). It has been suggested that media reporting of suicide can influence vulnerable individuals by raising awareness of suicide methods and normalising of suicidal behaviour (Goldney, 1989). In both Australia and New Zealand, guidelines have been introduced to encourage responsible reporting of suicide in the media (Hunter Institute of Mental Health, 2014; Ministry of Health, 1999). These include restrictions on disclosing details about methods, glamorising or sensationalising suicide and encouraging the inclusion of helplines in media reports.

Social factors that are more directly relevant to the individual are also powerful predictors of suicidality. In a comprehensive review, van Orden and colleagues (2010) argue that social isolation and a lack of social belonging are among the most powerful predictors of suicidal ideation and behaviour. They maintain that the sensation of being alone, without others to turn to, is a major factor in the development of suicidality.

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New Zealand–Australian television personality Charlotte Dawson, known for her roles on *Getaway* and *Australia's Next Top Model*, died from suicide in 2014. Researchers have shown that suicide rates increase in the month after a celebrity commits suicide. These findings provide evidence that the social environment is an influence on suicide.



## Psychological factors

Suicide may have many different meanings. Thoughts of suicide may arise from thinking one cannot cope with a situation, a desire to rejoin a deceased loved one, or a drive to escape from emotional pain or an emotional vacuum. Undoubtedly, the psychological variables involved in suicide vary across people, but many researchers have attempted to identify risk factors that operate across them.

Several researchers relate suicide to poor problem-solving skills (Linehan & Shearin, 1988). Problem-solving deficits do predict suicide attempts prospectively (Dieserud, Roysamb, Braverman, Dalgard, & Ekeberg, 2003). Deficits also relate to the seriousness of previous suicide attempts, even after controlling for severity of depression, age and intellectual functioning (Keilp et al., 2001).

A person who has trouble resolving problems can be expected to be more vulnerable to hopelessness. Hopelessness — the expectation that life will be no better in the future than it is in the present — is strongly tied to suicidality. High levels of hopelessness are associated with a fourfold elevation in the risk of suicide (Brown, Beck, Steer, & Grisham, 2000) and hopelessness is important even after controlling for depression levels (Beck, Kovacs, & Weissman, 1975).

### Virginia Woolf

On March 28, 1941, at the age of 59, Virginia Woolf drowned herself in the river near her Sussex home. Two suicide notes were found in the house, similar in content; one may have been written 10 days earlier, and it is possible that she may have made an unsuccessful attempt then, for she returned from a walk soaking wet, saying that she had fallen. The first was addressed to her sister Vanessa and the second to her husband, Leonard. To him, she wrote:

Dearest, I feel certain I am going mad again. I feel we can't go through another of those terrible times. And I shan't recover this time. I begin to hear voices, and I can't concentrate. So I am doing what seems the best thing to do. You have given me the greatest possible happiness. You have been in every way all that anyone could be. I don't think two people could have been happier till this terrible disease came. I can't fight it any longer. I know that I am spoiling your life, that without me you could work. And you will I know. You see I can't even write this properly. I can't read. What I want to say is I owe all the happiness of my life to you. You have been entirely patient with me and incredibly good. I want to say that — everybody knows it. If anybody could have saved me it would have been you. Everything has gone from me but the certainty of your goodness. I can't go on spoiling your life any longer. I don't think two people could have been happier than we have been. V.

Quoted from pp. 400–401, Briggs, J. (2005). *Virginia Woolf: An Inner Life*. Orlando, FL: Harcourt, Inc.

Beyond these risk factors (e.g., poor problem solving, hopelessness), positive qualities may motivate a person to live and help a clinician to build a case for choosing life (Malone et al., 2000). One line of research builds on the Reasons for Living Inventory (Linehan, Goodstein, Nielsen, & Chiles, 1983). Items on this inventory tap into what is important to the person, such as responsibility to family and concerns about children. People with more reasons to live tend to be less suicidal than those with few reasons to live (Ivanoff, Jang, Smyth, & Linehan, 1994).

Although many people think about suicide, relatively few engage in suicidal actions. Additional variables predict the switch from thinking about suicide to acting on those thoughts (van Orden, Witte, Gordon, Bender, & Joiner, 2008). Among people who are experiencing suicidal thoughts, hundreds of studies demonstrate that people who are more impulsive are more likely to attempt suicide or to die from suicide (Brezo, Paris, & Turecki, 2006). Intense distress and hopelessness might set off thoughts about suicide, but suicidal actions might be driven by other factors, such as impulsivity.

English novelist and critic Virginia Woolf (1882–1941)



## Preventing suicide

Many people worry that talking about suicide will make it more likely to happen. Rather, clinicians have learned that it is helpful to talk about suicide openly and matter-of-factly. Giving a person permission to talk about suicide may relieve a sense of isolation.

Because they are ambivalent about their suicidal intentions, most will communicate their intentions in some way. ‘The prototypical suicidal state is one in which a person cuts his or her throat, cries for help at the same time and is genuine in both of these acts . . . Individuals would be happy not to do it, if they didn’t have to’ (Shneidman, 1987, p. 170). Among those who attempt suicide but do not die, 80 percent report within the next two days that they are either glad to be alive or ambivalent about whether they want to die (Henriques, Wenzel, Brown, & Beck, 2005). This ambivalence gives the clinician an important foothold.

## Treating the associated psychological disorder

One approach to suicide prevention builds on our knowledge that most people who kill themselves are suffering from a psychological disorder. Thus when Beck’s cognitive approach successfully lessens a patient’s depression, that patient’s suicidal risk is also reduced. Marsha Linehan’s dialectical behaviour therapy with borderline personality disorder patients provides another example of a treatment that is designed for a specific disorder but also provides protection from suicide (see the chapter on personality and personality disorders).

Studies have found that medications for mood disorders reduce the risk of suicidality three- to four-fold (Angst et al., 2002). Specifically, lithium appears effective in suicide prevention for people with bipolar disorder (Cipriani, Pretty, Hawton, & Geddes, 2005). Among people who have been diagnosed with depressive disorders, ECT and antidepressants reduce suicidality (Bruce et al., 2004; Kellner et al., 2005). Risperidone, an antipsychotic medication, also appears to reduce the risk of suicide attempts among people with schizophrenia (Meltzer, 2003).

## Treating suicidality directly

Cognitive-behavioural approaches appear to be the most promising therapies for reducing suicidality (van der Sande, Buskens, Allart, van der Graaf, & van Engeland, 1997). These programs have been found to reduce the risk of a future attempt among those who have tried to kill themselves by 50 percent compared to treatment as usually offered in the community (Brown et al., 2005). They have also been found to reduce suicidal ideation (Joiner, Voelz, & Rudd, 2001). In a meta-analysis of 28 treatment trials, adults who received CBT reported less hopelessness, suicidal ideation and suicidal behaviour than those who received no treatment or other forms of treatment (Tarrier, Taylor, & Gooding, 2008).

Cognitive-behavioural treatments include a set of strategies to prevent suicide (Brown, Henriques, Ratto, & Beck, 2002). Therapists help clients understand the emotions and thoughts that trigger urges to commit suicide. Therapists work with clients to challenge their negative thoughts and to provide new ways to tolerate emotional distress. They also help clients solve problems they are facing. The goal is to improve problem solving and social support and thereby to reduce the feelings of hopelessness that often precede these episodes.

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Australian swimming champion Ian Thorpe revealed in his 2012 autobiography, *This is Me*, that his struggles with depression were so severe that at times he considered suicide.



Professional organisations such as the Australian Psychological Society charge their members with protecting people from suicide even if doing so requires breaking the confidentiality of the therapist–patient relationship. Therapists are expected to take reasonable precautions when they learn a patient is suicidal (Roy, 1995). One approach to keeping such patients alive is to hospitalise them as a short-term means of keeping them safe until they can begin to consider ways of improving their lives.

Some have argued against involuntary hospitalisations and other efforts to keep people from killing themselves, seeing this as a violation of the right to liberty to make one's own choices, even if those end in death (Szasz, 1999). Most people who are prevented from killing themselves are grateful afterwards for another chance at life. There are no easy answers here, but it is important to raise the questions.

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Melbourne's West Gate Bridge is a notorious hotspot for suicides. A review of coroners' reports and hospital records identified 62 cases of suicide between 1991 and 1998. Following the installation of safety barriers in 2009, the number of suicides was reduced by 85 percent.



### **Broader approaches to suicide prevention**

It is exceedingly difficult to do controlled research on suicide prevention since the base rates are so low and the ability to track suicide in a large population is limited. One approach to this issue has been to study suicide prevention within the military, where rates of suicide are much higher than in the general population, programs can be offered to the entire community and outcomes can be tracked carefully. In one study, researchers examined suicide rates in the Air Force before and after implementation of a comprehensive suicide prevention program. The program provided training for military leaders and for soldiers to encourage and destigmatise help seeking, to normalise the experience of distress and to promote effective coping with distress. Implementation of the program led to a 25 percent drop in rates of completed suicide (Knox et al., 2010). It would appear that prevention efforts can reduce suicide rates.

Beyond prevention programs that target high-risk individuals, public health approaches to suicide prevention try to change how easily people can access the means they might use to commit suicide.

For decades, the most common ovens in England relied on coal gas. Coal gas was cheap and widely available, but before it was burned, it released levels of carbon monoxide that were so high as to be deadly if one put one's head into a coal gas oven for even a couple of minutes. By the late 1950s, about 2500 suicides per year (almost half of the suicides in Britain) were committed in this manner. The British government phased out coal gas and by the 1970s, almost no coal gas ovens remained. Over the course of those years, the British suicide rate dropped by a third and has remained at this lower rate.

Public health researchers often emphasise that most suicides tend to be impulsive — people often make a suicide attempt very quickly without much forethought. The more difficult it is to execute a suicidal action, the less likely it is to occur. For this reason, strategies such as erecting a barrier on a bridge, so that people cannot jump off the bridge on a moment's impulse, can be highly successful (Beautrais, Gibb, Fergusson, Horwood, & Larkin, 2009).

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## SUMMARY

### **3.1 Describe the symptoms of depression and mania, the diagnostic criteria for depressive disorders and bipolar disorders, and the epidemiology of these disorders.**

The DSM-5 contains two broad types of mood disorders: depressive disorders and bipolar disorders. Depressive disorders include major depressive disorder (MDD) and persistent depressive disorder as well as two more recently recognised disorders: premenstrual dysphoric disorder and disruptive mood dysregulation disorder. Major depression is characterised by severe episodes lasting at least two weeks, whereas persistent depressive disorder is characterised by symptoms that last at least two years.

Bipolar disorders include bipolar I disorder, bipolar II disorder and cyclothymic disorder. Bipolar I disorder is diagnosed on the basis of a single lifetime manic episode and bipolar II disorder is diagnosed on the basis of hypomania and major depression. Cyclothymia is defined by frequent shifts between mild depressive and manic symptoms that last at least two years.

Specifiers of mood disorders are used to differentiate patterns of symptoms. Seasonal and rapid cycling specifiers are defined by the pattern of episodes over time; other specifiers are used to label the features of the current episode, including mood-congruent or mood-incongruent psychotic features, mixed features, catatonia, peripartum onset, with anxious distress, suicide risk severity and, for depression, melancholia. The seasonal specifier has been well validated.

MDD is one of the most common psychological disorders, whereas bipolar I disorder affects 1 percent or less of the population. MDD and persistent depressive disorder affect twice as many women as men. Two-thirds of people with MDD will experience another episode. Bipolar disorder is even more recurrent than MDD — about 50 percent of people with bipolar I disorder experience four or more episodes.

### **3.2 Discuss the genetic, neurobiological, social and psychological factors that contribute to the mood disorders.**

Bipolar disorder is highly heritable. Major depression is modestly heritable. A polymorphism of the serotonin transporter gene may increase risk for depression, particularly in the context of environmental adversity.

Neurotransmitter models have traditionally focused on serotonin, dopamine and norepinephrine. Current research focuses on receptor sensitivity of the serotonin and dopamine systems. Receptor sensitivity is often tested by manipulating the levels of neurotransmitters. Tryptophan depletion studies indicate that deficits in serotonin receptors are associated with depression and bipolar disorder. It also appears that depression may be related to diminished dopamine receptor sensitivity and that mania may be related to enhanced dopamine receptor sensitivity.

Neuroimaging studies suggest that depression and bipolar disorder are both associated with changes in regions of the brain that are involved in emotion. These changes seem consistent with a greater emotional reactivity (heightened activity of the amygdala) but less ability to regulate emotion (diminished activity of the dorsolateral prefrontal cortex and hippocampus, and greater activity of the anterior cingulate).

As noted, depression and bipolar disorder seem similar on many of these biological variables. One difference between the two conditions seems to be that depression appears to be related to lower activity of the striatum, whereas striatum activity appears to be high among those with bipolar disorder.

Depression and bipolar disorder are both related to poor regulation of cortisol when assessed using the dex/CRH test. Cortisol dysregulation also predicts a more severe course of mood symptoms over time.

Research strongly suggests that childhood adversity and recent life events can increase the risk for MDD. Because many people do not become depressed after a life event, researchers have studied diatheses that could explain vulnerability to life events.

The interpersonal diatheses for depression include low social support, high expressed emotion, high need for reassurance and poor social skills. Beyond social factors, psychological risk factors can help explain why some people become depressed. Evidence suggests that neuroticism, which involves frequent and intense negative affect, predicts the onset of depression. Cognitive factors include a negative schema; negative beliefs about the self, world and future; biases to attend to and recall negative rather than positive information; stable and global attributions for stressors that lead to hopelessness; and tendencies to ruminate. Prospective evidence supports each of these cognitive models.

Many of the variables that predict MDD also appear to predict depressive symptoms within bipolar disorder. Mania appears to be predicted by sleep deprivation and by life events involving goal attainment.

### **3.3 Identify the medication and psychological treatments of mood disorders, as well as the current views of electroconvulsive therapy.**

Many different treatments are available for depression. Cognitive–behavioural therapy (CBT), behavioural couples therapy and interpersonal psychotherapy (IPT) have received support, while third wave therapies such as mindfulness-based cognitive therapy (MBCT) are growing in popularity. ECT is effective, but there are concerns about cognitive side effects. The three forms of antidepressants have been found to be similarly effective; SSRIs have become more popular because they have fewer side effects than MAOIs and tricyclic antidepressants. CT appears to be as effective as antidepressant medication even in the treatment of severe MDD. IPT, though, does not appear to be as powerful as antidepressant medication for MDD.

Medication treatment is the first line of defence against bipolar disorder. The best-researched mood stabiliser is lithium, but anticonvulsants and antipsychotic medications are also effective mood stabilisers. Recent findings cast doubt on whether antidepressant medication is helpful in bipolar disorder. Some psychological treatments may help when offered as supplements to medications for the treatment of bipolar disorder. The best-validated approaches include psychoeducation, CBT and family-focused therapy (FFT). Interpersonal and social rhythm therapy (IPSRT) also fared well in the largest trial of psychological supplements to medications. These treatments appear particularly helpful in improving adherence to medication regimens and relieving depressive symptoms within bipolar disorder.

### **3.4 Explain the epidemiology of suicide, as well as the neurobiological, social and psychological risk factors associated with suicide.**

Suicide and thoughts of suicide may be tragic consequences of mood disorders and are also frequently linked to other mental illnesses. In Australia, approximately 10 per 100 000 deaths are due to intentional self-injury. Male gender, older age and Aboriginal or Torres Strait Islander ethnicity are associated with greater risk of death by suicide.

Genetic factors may explain about half of the variance in who will attempt suicide. Biological factors implicated in depression (e.g., cortisol dysregulation and serotonin dysfunction) have also been shown to be important predictors of suicidality. Social factors such as media coverage of deaths by suicide and social isolation have been shown to contribute to the development of suicidality. Psychological factors implicated in suicidality include poor problem-solving skills and hopelessness, with impulsivity said to be involved in the switch from thinking about suicide to acting on suicidal thoughts.

Treating mental illnesses associated with suicide, including depression and bipolar disorder, can effectively reduce risk. Both medications and psychological interventions have demonstrated efficacy. Specific forms of CBT act directly on suicidality, targeting emotions and thoughts that trigger urges to commit suicide. Individuals at great risk of suicide may need to be hospitalised for their own protection.

Broader approaches to suicide prevention may target social risk factors, for example, guidelines regulating media reporting of deaths by suicide. Programs have been developed to reduce suicide in specific at-risk populations, such as military veterans. Public health approaches attempt to change how easily people may access the means to die by suicide, such as by installing safety barriers on bridges.

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## KEY TERMS

**anterior cingulate** in the subcortical region of the brain, the anterior portion of the cingulate gyrus, stretching about the corpus callosum

**antidepressant** any drug that alleviates depression; also widely used to treat anxiety disorders

**attribution** the explanation a person has for why an event or behaviour has occurred

**attributional style** a person's habitual manner of assigning causes to behaviours and events

**behavioural couples therapy** clinical approach to depression in which a couple works to improve communication and satisfaction; more likely to relieve relationship distress than individual cognitive therapy

**bipolar I disorder** a diagnosis defined on the basis of at least one lifetime episode of mania. Most people with this disorder also experience episodes of major depression

**bipolar II disorder** a form of bipolar disorder, diagnosed in those who have experienced at least one major depressive episode and at least one episode of hypomania

**cognitive biases** tendencies to perceive events in a negative manner, for example, by attending to or remembering negative information more than positive information; hypothesised to be driven by underlying negative schemas

**Cushing's syndrome** an endocrine disorder usually affecting young women, produced by oversecretion of cortisone and marked by mood swings, irritability, agitation and physical disfigurement

**cyclothymic disorder** a form of bipolar disorder characterised by swings between elation and depression over at least a two-year period, but with moods not so severe as manic or major depressive episodes

**deep brain stimulation** a neurosurgical treatment in which electrodes are implanted into specific brain regions

**disruptive mood dysregulation disorder** a DSM-5 disorder defined by severe temper outbursts and observably irritable mood between outbursts in youth older than age six

**dorsolateral prefrontal cortex** a region of the prefrontal cortex involved in working memory, motor planning, organisation and regulation, and implicated in many psychopathologies

**episodic disorder** a condition, such as major depressive disorder, whose symptoms dissipate but that tends to recur

**expressed emotion (EE)** hostility, criticism and emotional overinvolvement directed from other people towards the patient, usually within a family

**flight of ideas** a symptom of mania that involves a rapid shift in conversation from one subject to another with only superficial associative connections

**hopelessness theory** cognitive theory of depression that began with learned helplessness theory, was modified to incorporate attributions and has been modified again to emphasise hopelessness — an expectation that desirable outcomes will not occur and that no available responses can change the situation

**hypomania** an extremely elevated or irritable mood accompanied by symptoms such as increased energy and decreased need for sleep, but without the significant functional impairment associated with mania

**interpersonal and social rhythm therapy (IPSRT)** a behavioural treatment for bipolar disorder that aims to stabilise mood by addressing disruptions in circadian rhythms (e.g., stressful life events, irregular sleep/wake times)

**lithium** a drug useful in treating both mania and depression in bipolar disorder

**major depressive disorder (MDD)** a disorder of individuals who have experienced episodes of depression but not of mania. Depression episodes are marked by sadness or loss of pleasure, accompanied by symptoms such as feelings of worthlessness and guilt; withdrawal from others; loss of sleep, appetite or sexual desire; and either lethargy or agitation

**mania** intense elation or irritability, accompanied by symptoms such as excessive talkativeness, rapid thoughts, distractibility, grandiose plans, heightened activity and insensitivity to the negative consequences of actions

**melancholic** subtype of major depressive disorder in which the individual is unable to feel better even momentarily when something good happens, regularly feels worse in the morning and awakens early and suffers a deepening of other symptoms of depression

**mindfulness-based cognitive therapy (MBCT)** a recent adaptation of cognitive therapy which draws on so-called 'third wave' techniques such as mindfulness meditation to help patients learn to see thoughts as harmless mental events, rather than challenge their content as in cognitive therapy

**monoamine oxidase inhibitors (MAOIs)** a group of antidepressant drugs that prevent the enzyme monoamine oxidase from deactivating catecholamines and indolamines

**mood disorders** disorders, such as depressive disorders or mania, in which there are disabling disturbances in emotion

**negative triad** in Beck's theory of depression, a person's negative views of the self, the world and the future, in a reciprocal causal relationship with pessimistic assumptions (schemas) and cognitive biases such as selective abstraction

**neuroticism** the tendency to react to events with greater-than-average negative affect; a strong predictor of onset of anxiety disorders and depression

**non-suicidal self-injury (NSSI)** behaviours that are meant to cause immediate bodily harm but are not intended to cause death

**peripartum onset** onset during pregnancy or within four weeks postpartum, characterising a subtype of episodes of major depressive disorder or mania

**persistent depressive disorder** a DSM-5 disorder defined by depressive symptoms that last at least two years

**premenstrual dysphoric disorder** a depressed, anxious, labile or irritable mood that reoccurs monthly during the luteal phase of the menstrual cycle. The symptoms are more distressing and disabling than the more commonly experienced premenstrual syndrome (PMS)

**psychoeducational approaches** especially with bipolar disorder and schizophrenia, the component of treatment that helps people learn about symptoms, expected time course, triggers for symptoms and treatment strategies

**psychomotor agitation** a symptom characterised by pacing, restlessness and inability to sit still

**psychomotor retardation** a symptom commonly observed in major depressive disorder in which the person moves his or her limbs and body slowly

**rapid cycling** term applied to bipolar disorders if the person has experienced at least four episodes within the past year

**reward system** system of brain structures involved in the motivation to pursue rewards. Believed to be involved in depression, mania, schizophrenia and substance use disorders

**rumination** repetitive thought about why a person is experiencing a negative mood

**seasonal affective disorder** a subtype of mood disorders in which episodes consistently occur at the same time of year; in the most common form, major depressive episodes consistently occur in the winter

**selective serotonin reuptake inhibitors (SSRIs)** a specific form of serotonin reuptake inhibitors (SRIs) with less effect on dopamine and norepinephrine levels. SSRIs inhibit the reuptake of serotonin into the presynaptic neuron, so that serotonin levels in the cleft are sustained for a longer period of time

**serotonin noradrenaline reuptake inhibitors (SNRIs)** SNRIs inhibit the reuptake of both serotonin and noradrenaline into the presynaptic neuron, so that levels of these neurotransmitters in the cleft are sustained for a longer period of time

**striatum** in the subcortical region of the brain; involved in the reward system

**suicide** the intentional taking of one's own life

**transcranial magnetic stimulation (rTMS)** a non-invasive technique in which pulsing magnets are used to intensify or diminish brain activity in a given region

**tricyclic antidepressants** a group of antidepressants with molecular structures characterised by three fused rings; they interfere with the reuptake of norepinephrine and serotonin

**tryptophan** amino acid that is the major precursor of serotonin. Experimental depletion has found that a lowered serotonin level causes temporary depressive symptoms in people with a personal or family history of depression

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## WEBSITES

1. The Black Dog Institute is a not-for-profit organisation involved in the research, clinical management and community promotion of mood disorders including depression and bipolar disorder. ([www.blackdoginstitute.org.au](http://www.blackdoginstitute.org.au))
2. Beyond Blue is an organisation that aims to challenge stigma and discrimination by providing support and information on depression, anxiety and suicide. ([www.beyondblue.com.au](http://www.beyondblue.com.au))
3. CREST.BD Bipolar Wellness Centre is an online educational resource centre containing information about living well with bipolar disorder. ([www.bdwellness.com](http://www.bdwellness.com))
4. MoodGym is an online cognitive behaviour therapy intervention teaching skills for coping with and preventing depression. (<https://moodgym.anu.edu.au/welcome>)

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## REFERENCES

- Abramson, L. Y., Metalsky, G. I., & Alloy, L. B. (1989). Hopelessness depression: A theory-based subtype of depression. *Psychological Review*, 96, 358–372.
- Alloy, L. B., Abramson, L. Y., Smith, J. M., Gibb, B. E., & Neeren, A. M. (2006). Role of parenting and maltreatment histories in unipolar and bipolar mood disorders: Mediation by cognitive vulnerability to depression. *Clinical Child and Family Psychology Review*, 9, 23–64.
- Alloy, L. B., Abramson, L. Y., Walshaw, P. D., Cogswell, A., Grandin, L. D., Hughes, M. E., et al. (2008). Behavioral approach system and behavioral inhibition system sensitivities and bipolar spectrum disorders: Prospective prediction of bipolar mood episodes. *Bipolar Disorders*, 10, 310–322.
- Altshuler, L. L., Kupka, R. W., Helleman, G., Frye, M. A., Sugar, C. A., McElroy, S. L., et al. (2010). Gender and depressive symptoms in 711 patients with bipolar disorder evaluated prospectively in the Stanley Foundation Bipolar Treatment Outcome Network. *American Journal of Psychiatry*, 167, 708–715.
- Anand, A., Verhoeff, P., Seneca, N., Zoghbi, S. S., Seibyl, J. P., Charney, D. S., et al. (2000). Brain SPECT imaging of amphetamine-induced dopamine release in euthymic bipolar disorder patients. *American Journal of Psychiatry*, 157, 1109–1114.
- Andrews, G., Cuijpers, P., Craske, M. G., McEvoy, P., & Titov, N. (2010). Computer therapy for the anxiety and depressive disorders is effective, acceptable and practical health care: A meta-analysis. *PloS One*, 5, e13196.
- Andrews, P. W., & Thomson, J. A. (2009). The bright side of being blue: Depression as an adaptation for analyzing complex problems. *Psychological Review*, 116, 620–654.
- Angst, F., Stassen, H. H., Clayton, P. J., & Angst, J. (2002). Mortality of patients with mood disorders: Follow-up over 34–38 years. *Journal of Affective Disorders*, 68, 167–181.
- Arias, E., Anderson, R. N., Kung, H. C., Murphy, S. L., & Kochanek, K. D. (2003). *Deaths: Final Reports*, 52. Hyattsville, MD: National Center for Health Statistics. DHHS Publication No. 2003–1120.
- Aubry, J., Gervasoni, N., Osiek, C., Perret, G., Rossier, M. F., Bertschy, G., et al. (2007). The DEX/CRH neuroendocrine test and the prediction of depressive relapse in remitted depressed outpatients. *Journal of Psychiatric Research*, 41, 290–294.
- Australian Bureau of Statistics (ABS). (2008). *National survey of mental health and wellbeing: summary of results, 2007*. Canberra: ABS.

- Australian Bureau of Statistics (ABS). (2016a). *Causes of death, Australia, 2015*. Canberra: ABS.
- Australian Bureau of Statistics. (2016b). *Patterns of use of mental health services and prescription medications, 2011*. Canberra: ABS.
- Australian Institute of Health and Welfare. (2016). *Australian burden of disease study: impact and causes of illness and deaths in Australia 2011*. Canberra: AIHW.
- Barbato, A., & D'Avanzo, B. (2008). Efficacy of couple therapy as a treatment for depression: A meta-analysis. *Psychiatric Quarterly*, 79, 121–132.
- Barth, J., Schumacher, M., & Herrmann-Lingen, C. (2004). Depression as a risk factor for mortality in patients with coronary heart disease: A meta-analysis. *Psychosomatic Medicine*, 66, 802–813.
- Beautrais, A. L., Gibb, S. J., Fergusson, D., Horwood, L. J., & Larkin, G. L. (2009). Removing bridge barriers stimulates suicides: An unfortunate natural experiment. *Australian and New Zealand Journal of Psychiatry*, 43, 495–497.
- Beck, A. T. (1967). *Depression: Clinical, experimental and theoretical aspects*. New York: Harper & Row.
- Beck, A. T., Kovacs, M., & Weissman, A. (1975). Hopelessness and suicidal behavior: An overview. *Journal of the American Medical Association*, 234, 1146–1149.
- Beevers, C. G., Lee, H. J., Wells, T. T., Ellis, A. J., & Telch, M. J. (2011). Association of predeployment gaze bias for emotion stimuli with later symptoms of PTSD and depression in soldiers deployed in Iraq. *American Journal of Psychiatry*, 168, 735–741.
- Benkelfat, C., Ellenbogen, M. A., Dean, P., Palmour, R. M., & Young, S. N. (1994). Mood-lowering effect of tryptophan depletion: Enhanced susceptibility in young men at genetic risk for major affective disorders. *Archives of General Psychiatry*, 51, 687–700.
- Bewernick, B. H., Hurlmann, R., Matusch, A., Kayser, S., Grubert, C., Hadrysiewicz, B., et al. (2010). Nucleus accumbens deep brain stimulation decreases ratings of depression and anxiety in treatment-resistant depression. *Biological Psychiatry*, 67, 110–116.
- Brems, C. (1995). Women and depression: A comprehensive analysis. In E. E. Beckham & W. Leber (Eds.), *Handbook of depression* (2nd ed., pp. 539–566). New York: Guilford Press.
- Brezo, J., Paris, J., & Turecki, G. (2006). Personality traits as correlates of suicidal ideation, suicide attempts, and suicide completions: A systematic review. *Acta Psychiatrica Scandinavica*, 113, 180–206.
- Briggs, J. (2005). *Virginia Woolf: An Inner Life*. Orlando, FL: Harcourt, Inc.
- Bromet, E., Andrade, L. H., Hwang, I., Sampson, N. A., Alonso, J., de Girolamo, G., et al. (2011). Cross-national epidemiology of DSM-IV major depressive episode. *BMC Medicine*, 9, 90.
- Brown, A. S., Schaefer, C. A., Quesenberry, C. P., Jr., Liu, L., Babulas, V. P., & Susser, E. S. (2005). Maternal exposure to toxoplasmosis and risk of schizophrenia in adult offspring. *American Journal of Psychiatry*, 162, 767–773.
- Brown, G. W., & Andrews, B. (1986). Social support and depression. In R. Trumbull & M. H. Appley (Eds.), *Dynamics of stress: Physiological, psychological, and social perspectives* (pp. 257–282). New York: Plenum.
- Brown, G. W., & Harris, T. O. (1989). Depression. In T. O. Harris & G. W. Brown (Eds.), *Life events and illness* (pp. 49–93). New York: Guilford Press.
- Brown, G. K., Beck, A. T., Steer, R. A., & Grisham, J. R. (2000). Risk factors for suicide in psychiatric outpatients: A 20-year prospective study. *Journal of Consulting and Clinical Psychology*, 68, 371–377.
- Brown, G. K., Henriques, G. R., Ratto, C., & Beck, A. T. (2002). *Cognitive therapy treatment manual for suicide attempters*. Philadelphia: University of Pennsylvania.
- Bruce, M. L., Ten Have, T. R., Reynolds III, C. F., Katz, I. I., Schulberg, H. C., Mulsant, B. H., et al. (2004). Reducing suicidal ideation and depressive symptoms in depressed older primary care patients. *Journal of the American Medical Association*, 291, 1081–1091.
- Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse: A meta-analysis. *Archives of General Psychiatry*, 55, 547–553.
- Carter, J. S., & Garber, J. (2011). Predictors of the first onset of a major depressive episode and changes in depressive symptoms across adolescence: stress and negative cognitions. *Journal of Abnormal Psychology*, 120, 779–796.
- Caspi, A., Sugden, K., Moffitt, T. E., Taylor, A., Craig, I. W., Harrington, H., et al. (2003). Influence of life stress on depression: Moderation by a polymorphism in the 5-HTT gene. *Science*, 301, 386–389.
- Caspi, A., Hariri, A. R., Holmes, A., Uher, R., & Moffitt, T. E. (2010). Genetic sensitivity to the environment: The case of the serotonin transporter gene and its implications for studying complex diseases and traits. *American Journal of Psychiatry*, 167, 509–527.
- Centers for Disease Control and Prevention. (2006). Homicides and suicides—National violent death reporting system, United States, 2003–2004. *American Journal of Medicine*, 296, 506–510.
- Chang, S. M., Hahm, B., Lee, J., Shin, M. S., Jeon, H. J., Hong, J., et al. (2008). Cross-national difference in the prevalence of depression caused by the diagnostic threshold. *Journal of Affective Disorders*, 106, 159–167.
- Chen, C. H., Suckling, J., Lennox, B. R., Ooi, C., & Bullmore, E. T. (2011). A quantitative meta-analysis of fMRI studies in bipolar disorder. *Bipolar Disorders*, 13, 1–15.
- Churchill, R., Moore, T. H. M., Furukawa, T. A., Caldwell, D. M., Davies, P., Jones, H., . . . Hunot, V. (2013). 'Third wave' cognitive and behavioural therapies versus treatment as usual for depression. *Cochrane Database of Systematic Reviews* (10).
- Cipriani, A., Pretty, H., Hawton, K., & Geddes, J. R. (2005). Lithium in the prevention of suicidal behavior and all-cause mortality in patients with mood disorders: A systematic review of randomized trials. *American Journal of Psychiatry*, 162, 1805–1819.

- Cole, D. A., Martin, J. M., Powers, B., & Truglio, R. (1990). Modeling causal relations between academic and social competence and depression: A multitrait-multimethod longitudinal study of children. *Journal of Abnormal Psychology, 105*, 258–270.
- Colom, F., Vieta, E., Reinares, M., Martinez-Aran, A., Torrent, C., Goikolea, J. M., & Gasto, C. (2003). Psychoeducation efficacy in bipolar disorders: Beyond compliance enhancement. *Journal of Clinical Psychiatry, 64*, 1101–1105.
- Colombo, C., Benedetti, F., Barbini, B., Campori, E., & Smeraldi, E. (1999). Rate of switch from depression into mania after therapeutic sleep deprivation in bipolar depression. *Psychiatry Research, 86*, 267–270.
- Cohen, P. (2008, February 21). Midlife suicide rises, puzzling researchers, *New York Times*, pp. 1–4.
- Coryell, W., & Schlesser, M. (2001). The dexamethasone suppression test and suicide prevention. *Archives of General Psychiatry, 158*, 748–753.
- Coyne, J. C. (1976). Depression and the response of others. *Journal of Abnormal Psychology, 85*, 186–193.
- Cross-Disorder Group of the Psychiatric Genomics Consortium. (2013). Identification of risk loci with shared effects on five major psychiatric disorders: a genome-wide analysis. *The Lancet, 381*(9875), 1371–1379.
- Cuellar, A. K., Johnson, S. L., & Winters, R. (2005). Distinctions between bipolar and unipolar depression. *Clinical Psychology Review, 25*, 307–339.
- Cuijpers, P., Cristea, I. A., Karyotaki, E., Reijnders, M., & Huibers, M. J. H. (2016). How effective are cognitive behaviour therapies for major depression and anxiety disorders? A meta-analytic update of the evidence. *World Psychiatry, 15*(3), 245–258.
- Daley, S. E., Hammen, C., & Rao, U. (2000). Predictors of first onset and recurrence of major depression in young women during the 5 years following high school graduation. *Journal of Abnormal Psychology, 109*, 525–533.
- Dantzer, R., O'Connor, J. C., Freund, G. G., Johnson, R. W., & Kelley, K. W. (2008). From inflammation to sickness and depression: when the immune system subjugates the brain. *Nature Reviews Neuroscience, 9*, 46–56.
- Davidson, R. J., Pizzagalli, D., & Nitschke, J. B. (2002). The representation and regulation of emotion in depression: Perspectives from affective neuroscience. In C. L. Hammen & I. H. Gotlib (Eds.), *Handbook of Depression* (pp. 219–244). New York: Guilford Press.
- Davila, J., Hammen, C. L., Burge, D., Paley, B., & Daley, S. E. (1995). Poor interpersonal problem solving as a mechanism of stress generation in depression among adolescent women. *Journal of Abnormal Psychology, 104*, 592–600.
- Depue, R. A., Collins, P. F., & Luciano, M. (1996). A model of neurobiology: Environment interaction in developmental psychopathology. In M. F. Lenzenweger & J. J. Haugaard (Eds.), *Frontiers of Developmental Psychopathology* (pp. 44–76). New York: Oxford University Press.
- Depue, R. A., & Iacono, W. G. (1989). Neurobehavioral aspects of affective disorders. *Annual Review of Psychology, 40*, 457–492.
- Dickerson, S. S., Gruenewald, T. L., & Kemeny, M. E. (2009). Psychobiological responses to social self threat: Functional or detrimental? *Self and Identity, 8*, 270–285.
- Dieserud, G., Roysamb, E., Braverman, M. T., Dalgard, O. S., & Ekeberg, O. (2003). Predicating repetition of suicide attempt: A prospective study of 50 suicide attempters. *Archives of Suicide Research, 7*, 1–15.
- DiLillo, D., Giuffrè, D., Tremblay, G. C., & Peterson, L. (2001). A closer look at the nature of intimate partner violence reported by women with a history of child sexual abuse. *Journal of Interpersonal Violence, 16*, 116–132.
- Disner, S. G., Beevers, C. G., Lee, H.-J., Ferrell, R. E., Hariri, A. R., & Telch, M. J. (2013). War zone stress interacts with the 5-HTTLPR polymorphism to predict the development of sustained attention for negative emotion stimuli in soldiers returning from Iraq. *Clinical Psychological Science, 1*, 413–425.
- Dobson, K. S., Hollon, S. D., Dimidjian, S., Schmalong, K. B., Kohlenberg, R. J., Gallop, R., et al. (2008). Randomized trial of behavioral activation, cognitive therapy, and antidepressant medication in the prevention of relapse and recurrence in major depression. *Journal of Consulting and Clinical Psychology, 76*, 468–477.
- Edvardsen, J., Torgersen, S., Roysamb, E., Lygren, S., Skre, I., Onstad, S., & Oien, P. A. (2008). Heritability of bipolar spectrum disorders. Unity or heterogeneity? *Journal of Affective Disorders, 106*, 229–240.
- Elkin, I., Shea, M. T., Watkins, J. T., Imber, S. D., Sotsky, S. M., Collins, J. F., et al. (1989). National Institute of Mental Health Treatment of Depression Collaborative Research Program. General effectiveness of treatments. *Archives of General Psychiatry, 46*, 971–982; discussion 983.
- Enserink, M. (1999). Drug therapies for depression: From MAO inhibitors to substance. *Science, 284*, 239.
- Fanous, A. H., Prescott, C. A., & Kendler, K. S. (2004). The prediction of thoughts of death or self-harm in a population-based sample of female twins. *Psychological Medicine, 34*, 301–312.
- Fletcher, K., Parker, G., Paterson, A., & Synnott, H. (2013). High-risk behaviour in hypomanic states. *Journal of Affective Disorders, 151*, 50–56.
- Food and Drug Administration. (2008, January 31). Serious health risks with antiepileptic drugs. Public Health Advisory. Retrieved from [www.fda.gov/consumer/updates/antiepileptic020508.html](http://www.fda.gov/consumer/updates/antiepileptic020508.html).
- Fournier, J. C., DeRubeis, R. J., Hollon, S. D., Dimidjian, S., Amsterdam, J. D., Shelton, R. C., & Fawcett, J. (2010). Antidepressant drug effects and depression severity: A patient-level meta-analysis. *Journal of the American Medical Association, 303*, 47–53.
- Frank, E., Kupfer, D. J., Perel, J. M., Cornes, C., Jarrett, D. B., Mallinger, A. G., et al. (1990). Three-year outcomes for maintenance therapies in recurrent depression. *Archives of General Psychiatry, 47*, 1093–1099.

- Frank, E., Swartz, H. A., & Kupfer, D. J. (2000). Interpersonal and social rhythm therapy: managing the chaos of bipolar disorder. *Biological Psychiatry*, 48(6), 593–604.
- Franklin, J. C., Hessel, E. T., Aaron, R. V., Arthur, M. S., Heilbron, N., & Prinstein, M. J. (2010). The functions of nonsuicidal self-injury: Support for cognitive-affective regulation and opponent processes from a novel psychophysiological paradigm. *Journal of Abnormal Psychology*, 119, 850–862.
- Fremouw, W. J., De Perczel, M., & Ellis, T. E. (1990). *Suicide risk: Assessment and response guidelines*. New York: Pergamon Press.
- Galante, J., Iribarren, S. J., & Pearce, P. F. (2013). Effects of mindfulness-based cognitive therapy on mental disorders: a systematic review and meta-analysis of randomised controlled trials. *Journal of Research in Nursing*, 18(2), 133–155.
- Gallo, C. L., & Pfeffer, C. R. (2003). Children and adolescents bereaved by a suicidal death: Implications for psychosocial outcomes and interventions. In A. Apter & R. A. King (Eds.), *Suicide in children and adolescents* (pp. 294–312). New York: Cambridge University Press.
- Geddes, J. R., Burgess, S., Hawton, K., Jamison, K., & Goodwin, G. M. (2004). Long-term lithium therapy for bipolar disorder: Systematic review and meta-analysis of randomized controlled trials. *American Journal of Psychiatry*, 161, 217–222.
- Geddes, J. R., Carney, S. M., Davies, C., Furukawa, T. A., Frank, E., Kupfer, D. J., et al. (2003). Relapse prevention with antidepressant drug treatment in depressive disorders: A systematic review. *The Lancet*, 361, 653–661.
- Goldapple, K., Segal, Z., Garson, C., Lau, M., Bieling, P., Kennedy, S., et al. (2004). Modulation of cortical-limbic pathways in major depression. *Archives of General Psychiatry*, 61, 34–41.
- Golden, R. N., Gaynes, B. N., Ekstrom, R. D., Hamer, R. M., Jacobsen, F. M., Suppes, et al. (2005). The efficacy of light therapy in the treatment of mood disorders: A review and meta-analysis of the evidence. *American Journal of Psychiatry*, 162, 656–662.
- Goldney, R. D. (1989). Suicide: the role of the media. *Australian and New Zealand Journal of Psychiatry*, 23(1), 30–34.
- Goodwin, F., & Jamison, K. (2007). *Manic-depressive illness: Bipolar disorders and recurrent depression* (2nd ed.). New York: Oxford University Press.
- Gotlib, H., & Joormann, J. (2010). Cognition and depression: Current status and future directions. *Annual Review of Clinical Psychology*, 6, 285–312.
- Gutman, D. A., & Nemeroff, C. B. (2003). Persistent central nervous system effects of an adverse early environment: Clinical and preclinical studies. *Physiology and Behavior*, 79, 471–478.
- Haaga, D. A. F., Dyck, M. J., & Ernst, D. (1991). Empirical status of cognitive theory of depression. *Psychological Bulletin*, 110, 215–236.
- Hamilton, J. P., Etkin, A., Furman, D. J., Lemus, M. G., Johnson, R. F., & Gotlib, I. H. (2012). Functional neuroimaging of major depressive disorder: A meta-analysis and new integration of base line activation and neural response data. *American Journal of Psychiatry*, 169, 693–703.
- Hammen, C., Hazel, N. A., Brennan, P. A., & Najman, J. (2012). Intergenerational transmission and continuity of stress and depression: Depressed women and their offspring in 20 years of follow-up. *Psychological Medicine*, 42, 931–942.
- Hankin, B. L., & Abramson, L. Y. (2001). Development of gender differences in depression: An elaborated cognitive vulnerability-transactional stress theory. *Psychological Bulletin*, 127, 773–796.
- Hankin, B. L., Mermelstein, R., & Roesch, L. (2007). Sex differences in adolescent depression: Stress exposure and reactivity models. *Child Development*, 78, 279–295.
- Hanson, J. L., Adluru, N., Chung, M. K., Alexander, A. L., Davidson, R. J., & Pollak, S. D. (2013). Early neglect is associated with alterations in white matter integrity and cognitive functioning. *Child Development*, 84, 1566–1578.
- Harkness, K. L., Bagby, R. M., & Kennedy, S. H. (2012). Childhood maltreatment and differential treatment response and recurrence in adult major depressive disorder. *Journal of Consulting and Clinical Psychology*, 80, 342–353.
- Harmer, C. J., Goodwin, G. M., & Cowen, P. J. (2009). Why do antidepressants take so long to work? A cognitive neuropsychological model of antidepressant drug action. *British Journal of Psychiatry*, 195, 102–108.
- Harris, R. (2009). *ACT made simple: an easy-to-read primer on acceptance and commitment therapy*. Oakland, CA: New Harbinger Publications.
- Harrow, M., Goldberg, J. F., Grossman, L. S., & Meltzer, H. Y. (1990). Outcome in manic disorders: A naturalistic follow-up study. *Archives of General Psychiatry*, 47, 665–671.
- Henriques, G., Wenzel, A., Brown, G. K., & Beck, A. T. (2005). Suicide attempter's reaction to survival as a risk factor for eventual suicide. *American Journal of Psychiatry*, 162, 2180–2182.
- Heuser, I., Yassouridis, A., & Holsboer, F. (1994). The combined dexamethasone CRH test: A refined laboratory test for psychiatric disorders. *Journal of Psychiatric Research*, 28, 341–346.
- Hibbeln, J. R., Nieminen, L. R. G., Blasbalg, T. L., Riggs, J. A., & Lands, W. E. M. (2006). Healthy intakes of n-3 and n-6 fatty acids: Estimations considering worldwide diversity. *Journal of Clinical Nutrition*, 83, 1483S–1493S.
- Hollon, S. D., DeRubeis, R. J., Shelton, R. C., Amsterdam, J. D., Salomon, R. M., & O'Reardon, J. P. (2005). Prevention of relapse following cognitive therapy vs medications in moderate to severe depression. *Archives of General Psychiatry*, 62, 417–422.
- Hollon, S. D., Thase, M. E., & Markowitz, J. C. (2002). Treatment and prevention of depression. *Psychological Science in the Public Interest*, 3, 39–77.
- Holtzheimer, P. E., & Mayberg, H. S. (2011). Deep brain stimulation for psychiatric disorders. *Annual Review of Neuroscience*, 34, 289–307.

- Houenou, J., Frommberger, J., Carde, S., Glasbrenner, M., Diener, C., Leboyer, M., & Wessa, M. (2011). Neuroimaging-based markers of bipolar disorder: Evidence from two meta-analyses. *Journal of Affective Disorders*, 132, 344–355.
- Hunter Institute of Mental Health. (2014). *Reporting suicide and mental illness: a mindframe resource for media professionals*. Newcastle: Hunter Institute of Mental Health.
- Ivanoff, A., Jang, S. J., Smyth, N. J., & Linehan, M. M. (1994). Fewer reasons for staying alive when you are thinking of killing yourself: The Brief Reasons for Living Inventory. *Journal of Psychopathology and Behavioral Assessment*, 16, 1–13.
- Jacobson, N. S., Dobson, K. S., Fruzzetti, A. E., & Schmaling, K. B. (1991). Marital therapy as a treatment for depression. *Journal of Consulting and Clinical Psychology*, 59, 547–557.
- Jacobson, N. S., & Gortner, E. T. (2000). Can depression be de-medicalized in the 21st century?: Scientific revolutions, counter-revolutions and the magnetic field of normal science. *Behaviour Research and Therapy*, 38, 103–117.
- Jamison, K. R. (1993). *Touched with fire: Manic-depressive illness and the artistic temperament*. New York: Simon & Schuster.
- Johnson, S. L., Cuellar, A. K., & Peckham, A. D. (Eds.). (2014). *Risk factors for bipolar disorder* (3rd ed.). New York: Guilford Press.
- Johnson, S. L., Edge, M. D., Holmes, M. K., & Carver, C. S. (2012). The behavioral activation system and mania. *Annual Review of Clinical Psychology*, 8, 243–267.
- Johnson, S. L., Murray, G., Fredrickson, B., Youngstrom, E. A., Hinshaw, S., Bass, J. M., . . . Salloum, I. (2012). Creativity and bipolar disorder: Touched by fire or burning with questions? *Clinical Psychology Review*, 32(1), 1–12.
- Joiner, T. E., Alfano, M. S., & Metalsky, G. I. (1992). When depression breeds contempt: Reassurance seeking, self-esteem, and rejection of depressed college students by their roommates. *Journal of Abnormal Psychology*, 101, 165–173.
- Joiner, T. E. (1995). The price of soliciting and receiving negative feedback: Self-verification theory as a vulnerability to depression theory. *Journal of Abnormal Psychology*, 104, 364–372.
- Joiner, T. E., Brown, J. S., & Wingate, L. R. (2005). The psychology and neurobiology of suicidal behavior. *Annual Review of Psychology*, 56, 287–314.
- Joiner, T. E., & Metalsky, G. I. (2001). Excessive reassurance-seeking: delineating a risk factor involved in the development of depressive symptoms. *Psychological Science*, 12, 371–378.
- Joiner, T. E., Voelz, Z. R., & Rudd, M. D. (2001). For suicidal young adults with comorbid depressive and anxiety disorders, problem-solving treatment may be better than treatment as usual. *Professional Psychology: Research and Practice*, 32, 278–282.
- Jorm, A. F., Christensen, H., Henderson, A. S., Jacomb, P. A., Korten, A. E., & Rodgers, B. (2000). Predicting anxiety and depression from personality: Is there a synergistic effect of neuroticism and extraversion? *Journal of Abnormal Psychology*, 109, 145–149.
- Judd, L. L. (1997). The clinical course of unipolar major depressive disorders. *Archives of General Psychiatry*, 54, 989–991.
- Judd, L. L., Akiskal, H. S., Maser, J. D., Zeller, P. J., Endicott, J., Coryell, W., et al. (1998). A prospective 12-year study of subsyndromal and syndromal depressive symptoms in unipolar major depressive disorders. *Archives of General Psychiatry*, 55, 694–701.
- Just, N., & Alloy, L. B. (1997). The response styles theory of depression: Tests and an extension of the theory. *Journal of Abnormal Psychology*, 106, 221–229.
- Karg, K. (2011). The serotonin transporter promoter variant (5-HTTLPR), stress, and depression meta-analysis revisited: Evidence of genetic moderation. *Archives of General Psychiatry*, 68, 444.
- Keilp, J. G., Sackeim, H. A., Brodsky, B. S., Oquendo, M. A., Malone, K. M., & Mann, J. J. (2001). Neuropsychological dysfunction in depressed suicide attempters. *Archives of General Psychiatry*, 158, 735–741.
- Keller, M. B., McCullough, J. P., Klein, D. N., Arnow, B., Dunner, D. L., Gelenberg, A. J., et al. (2000). A comparison of nefazodone, the cognitive behavioral-analysis system of psychotherapy, and their combination for the treatment of chronic depression. *The New England Journal of Medicine*, 342, 1462–1470.
- Kellner, C. H., Fink, M., Knapp, R., Petrides, G., Husain, M., Rummans, T., et al. (2005). Relief of expressed suicidal intent by ECT: A consortium for research in ECT study. *American Journal of Psychiatry*, 162, 977–982.
- Keltner, D., & Kring, A. M. (1998). Emotion, social function, and psychopathology. *Review of General Psychology*, 2, 320–342.
- Kendall, P. C., & Ingram, R. E. (1989). Cognitive-behavioral perspectives: Theory and research on depression and anxiety. In D. Watson & P. C. Kendall (Eds.), *Personality, psychopathology, and psychotherapy* (pp. 27–53). San Diego, CA: Academic Press.
- Kendler, K. S. (1997). The diagnostic validity of melancholic major depression in a population-based sample of female twins. *Archives of General Psychiatry*, 54, 299–304.
- Kendler, K. S., Hettema, J. M., Butera, F., Gardner, C. O., & Prescott, C. A. (2003). Life event dimensions of loss, humiliation, entrapment, and danger in the prediction of onsets of major depression and generalized anxiety. *Archives of General Psychiatry*, 60, 789–796.
- Kendler, K. S., Jacobson, K. C., Prescott, C. A., & Neale, M. C. (2003). Specificity of genetic and environmental risk factors for use and abuse/dependence of *Cannabis*, cocaine, hallucinogens, sedatives, stimulants, and opiates in male twins. *American Journal of Psychiatry*, 160, 687–695.
- Kendler, K. S., Myers, J., & Zisook, S. (2008). Does bereavement-related major depression differ from major depression associated with other stressful life events? *American Journal of Psychiatry*, 165, 1449–1455.
- Kessler, R. C. (2003). Epidemiology of women and depression. *Journal of Affective Disorders*, 74, 5–13.
- Kessler, R. C., Akiskal, H. S., Angst, J., Guyer, M., Hirschfeld, R. M. A., Merikangas, K. R., & Stang, P. E. (2006). Validity of the assessment of bipolar spectrum disorders in the WHO CIDI 3.0. *Journal of Affective Disorders*, 96, 259–269.

- Kessler, R. C., Berglund, P., Borges, G., Nock, M., & Wang, P. S. (2005). Trends in suicide ideation, plans, gestures, and attempts in the United States, 1990–1992 to 2001–2003. *Journal of the American Medical Association*, 293, 2487–2495.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Koretz, D., Merikangas, K. R., et al. (2003). The epidemiology of major depressive disorder: Results from the National Comorbidity Survey Replication (NCS-R). *Journal of the American Medical Association*, 289, 3095–3105.
- Kessler, R. C., Berglund, P., Demler, O., Jin, R., Merikangas, K. R., & Walters, E. E. (2005). Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the national comorbidity survey replication. *Archives of General Psychiatry*, 62, 593–602.
- Kessler, R. C., Birnbaum, H. G., Shahly, V., Bromet, E., Hwang, I., McLaughlin, K. A., et al. (2010). Age differences in the prevalence and co-morbidity of DSM-IV major depressive episodes: results from the WHO World Mental Health Survey Initiative. *Depression and Anxiety*, 27, 351–364.
- Kieseppa, T., Partonen, T., Haukka, J., Kaprio, J., & Lonnqvist, J. (2004). High concordance of bipolar I disorder in a nationwide sample of twins. *American Journal of Psychiatry*, 161, 1814–1821.
- Klein, D. N., Arnow, B. A., Barkin, J. L., Dowling, F., Kocsis, J. H., Leon, A. C., et al. (2009). Early adversity in chronic depression: Clinical correlates and response to pharmacotherapy. *Depression and Anxiety*, 26, 701–710.
- Klein, D. N., Schwartz, J. E., Rose, S., & Leader, J. B. (2000). Five-year course and outcome of dysthymic disorder: A prospective, naturalistic follow-up study. *American Journal of Psychiatry*, 157, 931–939.
- Klein, D. N., Shankman, S. A. M. A., & Rose, S. M. A. (2006). Ten-year prospective follow-up study of the naturalistic course of dysthymic disorder and double depression. *American Journal of Psychiatry*, 163, 872–880.
- Klerman, G. L. (1988). Depression and related disorders of mood (affective disorders). In A. M. Nicholi, Jr. (Ed.), *The new Harvard guide to psychiatry*. Cambridge, MA: Harvard University Press.
- Klerman, G. L., Weissman, M. M., Rounsaville, B. J., & Chevron, E. S. (1984). *Interpersonal psychotherapy of depression*. New York: Basic Books.
- Knox, K. L., Pflanz, S., Talcott, G. W., Campise, R. L., Lavigne, J. E., Bajorska, A., et al. (2010). The US Air Force suicide prevention program: Implications for public health policy. *American Journal of Public Health*, 100, 2457–2463.
- Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking “big” personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin*, 136(5), 768–821.
- Krishnan, V., & Nestler, E. J. (2010). Linking molecules to mood: New insight into the biology of depression. *American Journal of Psychiatry*, 167, 1305–1320.
- Kupfer, D. J. (2005). The increasing medical burden in bipolar disorder. *Journal of the American Medical Association*, 293, 2528–2530.
- Lam, D. H., Bright, J., Jones, S., Hayward, P., Schuck, N., Chisholm, D., et al. (2000). Cognitive therapy for bipolar illness—a pilot study of relapse prevention. *Cognitive Therapy and Research*, 24, 503–520.
- Lam, R. W., Levitt, A. J., Levitan, R. D., Enns, M. W., Morehouse, R., Michalak, E. E., et al. (2006). The Can-SAD study: A randomized controlled trial of the effectiveness of light therapy and fluoxetine in patients with winter seasonal affective disorder. *American Journal of Psychiatry*, 163, 805–812.
- LaMontagne, A. D. (2010). *Estimating the economic benefits of eliminating job strain as a risk factor for depression: full report*. Melbourne: Victorian Health Promotion Foundation (VicHealth).
- Leahy, R. L. (2003). *Cognitive therapy techniques: A practitioner's guide*. New York: Guilford Press.
- Levav, I., Kohn, R., Golding, J. M., & Weissman, M. M. (1997). Vulnerability of Jews to major depression. *American Journal of Psychiatry*, 154, 941–947.
- Lewinsohn, P. M. (Ed.). (1974). *A behavioral approach to depression*. New York: Springer Publishing.
- Lewinsohn, P. M., Joiner, T. E., & Rohde, P. (2001). Evaluation of cognitive diathesis-stress models in predicting major depressive disorder. *Journal of Abnormal Psychology*, 110, 203–215.
- Lieverse, R., Van Someren, E. J. W., Nielen, M. M. A., Uitdehaag, B. M. J., Smit, J. H., & Hoogendijk, W. J. (2011). Bright light treatment in elderly patients with nonseasonal major depressive disorder: A randomized placebo-controlled trial. *Archives of General Psychiatry*, 68, 61–70.
- Lin, E. H., Von Korff, M., Katon, W., Bush, T., Simon, G. E., Walker, E., & Robinson, P. (1995). The role of the primary care physician in patients' adherence to antidepressant therapy. *Medical Care*, 33(1), 67–74.
- Linehan, M. M. (1997). Behavioral treatments of suicidal behaviors: Definitional obfuscation and treatment outcomes. In D. M. Stoff & J. J. Mann (Eds.), *Neurobiology of suicide* (pp. 302–327). New York: Annals of the New York Academy of Sciences.
- Linehan, M. M., & Shearin, E. N. (1988). Lethal stress: A social-behavioral model of suicidal behavior. In S. Fisher & J. Reason (Eds.), *Handbook of life stress, cognition, and health*. New York: John Wiley & Sons.
- Linehan, M. M., Goodstein, J. L., Nielsen, S. L., & Chiles, J. A. (1983). Reasons for staying alive when you are thinking of killing yourself. *Journal of Consulting and Clinical Psychology*, 51, 276–286.
- Lumley, M. N., & Harkness, K. L. (2007). Specificity in the relations among childhood adversity, early maladaptive schemas, and symptom profiles in adolescent depression. *Cognitive Therapy and Research*, 31, 639–657.
- Luo, F., Florence, C. S., Quispe-Agnoli, M., Ouyang, L., & Crosby, A. E. (2011). Impact of business cycles on US suicide rates, 1928–2007. *American Journal of Public Health*, 101, 1139–1146.
- Malhi, G. S., Bassett, D., Boyce, P., Bryant, R., Fitzgerald, P. B., Fritz, K., . . . Singh, A. B. (2015). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for mood disorders. *Australian and New Zealand Journal of Psychiatry*, 49(12), 1087–1206.

- Malkoff-Schwartz, S., Frank, E., Anderson, B. P., Hlastala, S. A., Luther, J. F., Sherrill, J. T., et al. (2000). Social rhythm disruption and stressful life events in the onset of bipolar and unipolar episodes. *Psychological Medicine*, 30, 1005–1016.
- Malone, K. M., Oquendo, M. A., Haas, G. L., Ellis, S. P., Li, S., & Mann, J. J. (2000). Protective factors against suicidal acts in major depression: Reasons for living. *American Journal of Psychiatry*, 157, 1084–1088.
- Mann, J. J., Huang, Y. Y., Underwood, M. D., Kassir, S. A., Oppenheim, S., & Kelly, T. M. (2000). A serotonin transporter gene promoter polymorphism (5-HTTLPR) and prefrontal cortical binding in major depression and suicide. *Archives of General Psychiatry*, 57, 729–738.
- Martell, C. R., Addis, M. E., & Jacobson, N. S. (2001). *Ending depression one step at a time: The new behavioral activation approach to getting your life back*. New York: Oxford University Press.
- Mathews, A., & MacLeod, C. (2002). Induced processing biases have causal effects on anxiety. *Cognition and Emotion*, 16, 331–354.
- Mayberg, H. S., Lozano, A. M., Voon, V., McNeely, H. E., Seminowicz, D., Hamani, C., et al. (2005). Deep brain stimulation for treatment-resistant depression. *Neuron*, 45, 651–660.
- McCall, W. V., Reboussin, D. M., Weiner, R. D., & Sackeim, H. A. (2000). Titrated moderately suprathreshold vs fixed high-dose right unilateral electroconvulsive therapy: Acute antidepressant and cognitive effects. *Archives of General Psychiatry*, 57, 438–444.
- McCullough, J. P., Klein, D. N., Keller, M. B., Holzer, C. E., Davis, S. M., Korenstein, S. G., et al. (2000). Comparison of DSM-III major depression and major depression superimposed on dysthymia (double depression): Validity of the distinction. *Journal of Abnormal Psychology*, 109, 419–427.
- Meltzer, H. Y. (2003). Suicide in schizophrenia. *Journal of Clinical Psychiatry*, 64, 1122–1125.
- Merikangas, K. R., Akiskal, H. S., Angst, J., Greenberg, P. E., Hirschfeld, R. M. A., Petukhova, M., et al. (2007). Lifetime and 12-month prevalence of bipolar spectrum disorder in the National Comorbidity Survey Replication. *Archives of General Psychiatry*, 64, 543–552.
- Merikangas, K. R., Jin, R., He, J., Kessler, R. C., Lee, S., Sampson, N. A., et al. (2011). Prevalence and correlates of bipolar spectrum disorder in the World Mental Health Survey Initiative. *Archives of General Psychiatry*, 68, 241–251.
- Metalsky, G. I., Joiner, T. E., Hardin, T. S., & Abramson, L. Y. (1993). Depressive reactions to failure in a natural setting: A test of the hopelessness and self-esteem theories of depression. *Journal of Abnormal Psychology*, 102, 101–109.
- Meyer, B., Johnson, S. L., & Winters, R. (2001). Responsiveness to threat and incentive in bipolar disorder: Relations of the BIS/BAS scales with symptoms. *Journal of Psychopathology and Behavioral Assessment*, 23, 133–143.
- Miklowitz, D. J. (2008). Adjunctive psychotherapy for bipolar disorder: state of the evidence. *American Journal of Psychiatry*, 165(11), 1408–1419.
- Miklowitz, D. J., & Goldstein, M. J. (1997). *Bipolar disorder: A family-focused treatment approach*. New York: Guilford Press.
- Miklowitz, D. J., Otto, M. W., Frank, E., Reilly-Harrington, N. A., Wisniewski, S. R., Kogan, J. N., et al. (2007). Psychosocial treatments for bipolar depression: A 1-year randomized trial from the systematic treatment enhancement program. *Archives of General Psychiatry*, 64, 419–427.
- Miklowitz, D. J., Simoneau, T. L., Sachs-Ericsson, N., Warner, R., & Suddath, R. (1996). Family risk indicators in the course of bipolar affective disorder. In E. Mundt et al. (Ed.), *Interpersonal factors in the origin and course of affective disorders* (pp. 204–217). London: Gaskell.
- Miller, M., & Hemenway, D. (1999). The relationship between firearms and suicide: A review of the literature. *Aggression and Violent Behavior*, 4, 59–75.
- Ministry of Health. (1999). *Suicide and the media: the reporting and portrayal of suicide in the media. A Resource*. Wellington: Ministry of Health.
- Moffitt, T. E., Caspi, A., Harrington, H., Milne, B. J., Melchior, M., Goldberg, D., et al. (2007). Generalized anxiety disorder and depression: childhood risk factors in a birth cohort followed to age 32. *Psychological Medicine*, 37, 441–452.
- Morris, R. K., Faizal, M. A., Jones, A. P., Williamson, P. R., Bolton, C., & McCarthy, J. P. (2007). Interventions for helping people recognise early signs of recurrence in bipolar disorder. *Cochrane Database of Systematic Reviews*, Issue 1, CD004854.
- Morrow, J., & Nolen-Hoeksema, S. (1990). Effects of responses to depression on the remediation of depressive affect. *Journal of Personality and Social Psychology*, 58, 519–527.
- Murray, C. J. L., & Lopez, A. D. (1996). *The global burden of disease: A comprehensive assessment of disease, injuries, and risk factors in 1990 and projected to 2020*. Cambridge, MA: Harvard University Press.
- Murray, G. (2004). How common is seasonal affective disorder in temperate Australia? A comparison of BDI and SPAQ estimates. *Journal of Affective Disorders*, 81(1), 23–28.
- Murray, G., & Harvey, A. (2010). Circadian rhythms and sleep in bipolar disorder. In L. N. Yatham & M. Maj (Eds.), *Bipolar disorder: Clinical and neurobiological foundations* (pp. 263–274). New York: John Wiley & Sons.
- Murray, G., Leitan, N. D., Berk, M., Thomas, N., Michalak, E., Berk, L., . . . Kyrios, M. (2015). Online mindfulness-based intervention for late-stage bipolar disorder: pilot evidence for feasibility and effectiveness. *Journal of Affective Disorders*, 178, 46–51. doi: 10.1016/j.jad.2015.02.024
- Murray, G., & Michalak, E. E. (2012). The quality of life construct in bipolar disorder research and practice: Past, present and possible futures. *Bipolar Disorders*, 14(8), 793–796.

- Mykletun, A., Bjerkeset, O., Overland, S., Prince, M., Dewey, M., & Stewart, R. (2009). Levels of anxiety and depression as predictors of mortality: The HUNT study. *British Journal of Psychiatry*, 195, 118–125.
- Naranjo, C. A., Tremblay, L. K., & Busto, U. E. (2001). The role of the brain reward system in depression. *Progress in Neuro-Psychopharmacology and Biological Psychiatry*, 25, 781–823.
- Nelson, J. C. (2006). The STAR\*D study: A four-course meal that leaves us wanting more. *American Journal of Psychiatry*, 163, 1864–1866.
- Nemeroff, C. B., & Schatzberg, A. F. (1998). Pharmacological treatment of unipolar depression. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 212–225). New York: Oxford University Press.
- Neumeister, A., Konstantinidis, A., Stastny, J., Schwarz, M. J., Vitouch, O., Willeit, M., et al. (2002). Association between the serotonin transporter gene promoter polymorphism (5-HTTLPR) and behavioral responses to tryptophan depletion in healthy women with and without family history of depression. *Archives of General Psychiatry*, 59, 613–620.
- Nicholson, A., Kuper, H., & Hemingway, H. (2006). Depression as an aetiological and prognostic factor in coronary heart disease: A meta-analysis of 6362 events among 146538 participants in 54 observational studies. *European Heart Journal*, 27, 2763–2774.
- Nobler, M. S., Oquendo, M. A., Kegeles, L. S., Malone, K. M., Campbell, C., Sackheim, H. A., et al. (2001). Decreased regional brain metabolism after ECT. *American Journal of Psychiatry*, 158, 305–308.
- Nock, M. K. (2009). Why do people hurt themselves? New insights into the nature and function of self-injury. *Current Directions in Psychological Science*, 18, 78–83.
- Nock, M. K. (2010). Self-injury. *Annual Review of Clinical Psychology*, 6, 339–363.
- Nock, M. K., & Mendes, W. B. (2008). Physiological arousal, distress tolerance, and social problem-solving deficits among adolescent self-injurers. *Journal of Consulting and Clinical Psychology*, 76, 28–38.
- Nock, M. K., & Prinstein, M. J. (2004). A functional approach to the assessment of self-mutilative behavior. *Journal of Consulting and Clinical Psychology*, 72, 885–890.
- Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2006). Prevalence, subtypes, and correlates of DSM-IV conduct disorder in the National Comorbidity Survey Replication. *Psychological Medicine*, 36, 699–710.
- Nock, M. K., Prinstein, M. J., & Sterba, S. K. (2009). Revealing the form and functions of self-injurious thoughts and behaviors: A real-time ecological assessment study among adolescents and young adults. *Journal of Abnormal Psychology*, 118, 816–827.
- Nolen-Hoeksema, S. (1991). Responses to depression and their effects on the duration of depressive episodes. *Journal of Abnormal Psychology*, 100, 569–582.
- Nolen-Hoeksema, S. (2000). The role of rumination in depressive disorders and mixed anxiety/depressive symptoms. *Journal of Abnormal Psychology*, 109, 504–511.
- Nolen-Hoeksema, S. (2001). Gender differences in depression. *Current Directions in Psychological Science*, 10, 173–176.
- Osby, U., Brandt, L., Correia, N., Ekblom, A., & Sparen, P. (2001). Excess mortality in bipolar and unipolar disorder in Sweden. *Archives of General Psychiatry*, 58, 844–850.
- Pacchiarotti, I., Bond, D. J., Baldessarini, R. J., Nolen, W. A., Grunze, H., Licht, R. W., et al. (2013). The International Society for Bipolar Disorders (ISBD) task force report on antidepressant use in bipolar disorders. *American Journal of Psychiatry*, 170, 1249–1262.
- Palmer, B. A., Pankratz, V., & Bostwick, J. (2005). The lifetime risk of suicide in schizophrenia: A reexamination. *Archives of General Psychiatry*, 62, 247–253.
- Phillips, D. P. (1974). The influence of suggestion on suicide: Substantive and theoretical implications of the Werther effect. *American Sociological Review*, 39, 340–354.
- Phillips, D. P. (1985). The Werther effect. *The Sciences*, 25, 33–39.
- Phillips, M. L., Ladouceur, C. D., & Drevets, W. C. (2008). A neural model of voluntary and automatic emotion regulation: Implications for understanding the pathophysiology and neurodevelopment of bipolar disorder. *Molecular Psychiatry*, 13, 833–857.
- Pilhatsch, M., Vetter, N. C., Hubner, T., Ripke, S., Muller, K. U., Marxen, M., et al. (2014). Amygdala-function perturbations in healthy mid-adolescents with familial liability for depression. *Journal of the American Academy of Child & Adolescent Psychiatry*, 53, 559–558.
- Pizzagalli, D. A., Goetz, E., Ostacher, M., Iosifescu, D. V., & Perlis, R. H. (2008). Euthymic patients with bipolar disorder show decreased reward learning in a probabilistic reward task. *Biological Psychiatry*, 64, 162–168.
- Prien, R. F., & Potter, W. Z. (1993). Maintenance treatment for mood disorders. In D. L. Dunner (Ed.), *Current psychiatric therapy*. Philadelphia: Saunders.
- Raison, C. L., Capuron, L., & Miller, A. H. (2006). Cytokines sing the blues: Inflammation and the pathogenesis of depression. *Trends in Immunology*, 27, 24–31.
- Regeer, E. J., ten Have, M., Rosso, M. L., van Roijen, L. H., Vollebergh, W., & Nolen, W. A. (2004). Prevalence of bipolar disorder in the general population: A reappraisal study of the Netherlands Mental Health Survey and incidence study. *Acta Psychiatrica Scandinavica*, 110, 374–382.
- Regier, D. A., Narrow, W. E., Rae, D. S., & Manderscheid, R. W. (1993). The de facto US mental and addictive disorders service system: Epidemiologic catchment area prospective 1-year prevalence rates of disorders and services. *Archives of General Psychiatry*, 50, 85–94.
- Reilly-Harrington, N. A., Alloy, L. B., Fresco, D. M., & Whitehouse, W. G. (1999). Cognitive styles and life events interact to predict bipolar and unipolar symptomatology. *Journal of Abnormal Psychology*, 108, 567–578.

- Richards, R. L., Kinney, D. K., Lunde, I., Benet, M., & Merzel, A. (1988). Creativity in manic-depressives, cyclothymes, their normal relatives, and control subjects. *Journal of Abnormal Psychology*, 97, 281–288.
- Robinson, N. S., Garber, J., & Hillsman, R. (1995). Cognitions and stress: Direct and moderating effects on depression versus externalizing symptoms during the junior high school transition. *Journal of Abnormal Psychology*, 104, 453–463.
- Roecklein, K., Wong, P., Ernecoff, N., Miller, M., Donofry, S., Kamarck, M., Wood-Vasey, W. M., & Franzen, P. (2013). The post illumination pupil response is reduced in seasonal affective disorder. *Psychiatry Research*, 210, 150–158.
- Rohan, K. J., Roecklein, K. A., Tierney Lindsey, K., Johnson, L. G., Lippy, R. D., Lacy, T. J., et al. (2007). A randomized controlled trial of cognitive-behavioral therapy, light therapy, and their combination for seasonal affective disorder. *Journal of Consulting and Clinical Psychology*, 75, 489–500.
- Rosen, L. N., Targum, S. D., Terman, M., Bryant, M. J., Hoffman, H., Kasper, S. F., et al. (1990). Prevalence of seasonal affective disorder at four latitudes. *Psychiatry Research*, 31, 131–144.
- Roy, A. (1994). Recent biologic studies on suicide. *Suicide and life threatening behaviors*, 24, 10–24.
- Roy, A. (1995). *Suicide*. In H. I. Kaplan & B. J. Sadock (Eds.), *Comprehensive textbook of psychiatry* (pp. 1739–1752). Baltimore: Williams & Wilkins.
- Rush, A. J., Trivedi, M., Wisniewski, S. R., Nierenberg, A. A., Stewart, J. W., Warden, D., et al. (2006). Acute and longer-term outcomes in depressed outpatients requiring one or several treatment steps: A STARD report. *American Journal of Psychiatry*, 163, 1905–1917.
- Sachs, G. S., & Thase, M. E. (2000). Bipolar disorder therapeutics: Maintenance treatment. *Biological Psychiatry*, 48, 573–581.
- Sackeim, H. A., & Lisanby, S. H. (Eds.). (2001). *Physical treatments in psychiatry: Advances in electroconvulsive therapy, transcranial magnetic stimulation, and vagus nerve stimulation*. Washington, D. C.: American Psychiatric Publishing.
- Sackeim, H. A., Prudic, J., Fuller, R., Keilp, J., Lavori, P. W., & Olfson, M. (2007). The cognitive effects of electroconvulsive therapy in community settings. *Neuropsychopharmacology*, 32, 244–254.
- Salamone, J. D., & Correa, M. (2012). The mysterious motivational functions of mesolimbic dopamine. *Neuron*, 76, 470–485.
- Schaefer, H. S., Putnam, K. M., Benca, R. M., & Davidson, R. J. (2006). Event-related functional magnetic resonance imaging measures of neural activity to positive social stimuli in pre- and post-treatment depression. *Biological Psychiatry*, 60, 974–986.
- Scherk, H., Pajonk, F. G., & Leucht, S. (2007). Second-generation antipsychotic agents in the treatment of acute mania: A systematic review and meta-analysis of randomized controlled trials. *Archives of General Psychiatry*, 64, 442–455.
- Schmidt, N. B., Woolaway-Bickel, K., & Bates, M. (2000). Suicide and panic disorder: Integration of the literature and new findings. In M. D. Rudd & T. E. Joiner (Eds.), *Suicide science: Expanding the boundaries* (pp. 117–136). New York: Kluwer Academic/Plenum.
- Schneck, C. D., Miklowitz, D. J., Miyahara, S., Araga, M., Wisniewski, S. R., Gyulai, L., et al. (2008). The prospective course of rapid-cycling bipolar disorder: Findings from the STEP-BD. *American Journal of Psychiatry*, 165, 370–376.
- Seedat, S., Scott, K. M., Angermeyer, M., Berglund, P., Bromet, E., Brugha, T., et al. (2009). Cross-national associations between gender and mental disorders in the world health organization world mental health surveys. *Archives of General Psychiatry*, 66, 785–795.
- Segal, Z. V., Kennedy, S., Gemar, M., Hood, K., Pedersen, R., & Buis, T. (2006). Cognitive reactivity to sad mood provocation and the prediction of depressive relapse. *Archives of General Psychiatry*, 63, 749–755.
- Sheline, Y., Barch, D., Donnelly, J. M., Ollinger, J. M., Snyder, A. Z., & Mintun, M. A. (2001). Increased amygdala response to masked emotional faces in depressed subjects resolves with antidepressant treatment: An fMRI study. *Biological Psychiatry*, 50, 651–658.
- Shneidman, E. S. (1987). A psychological approach to suicide. In G. R. VandenBos & B. K. Bryant (Eds.), *Cataclysms, crises, and catastrophes: Psychology in action*. Washington, DC: American Psychological Association.
- Simon, G. E., Goldberg, D. P., Von Korff, M., & Ustun, T. B. (2002). Understanding cross-national differences in depression prevalence. *Psychological Medicine*, 32, 585–594.
- Simon, G. E., Von Korff, M., Piccinelli, M., Fullerton, C., & Ormel, J. (1999). An international study of the relation between somatic symptoms and depression. *New England Journal of Medicine*, 341, 1329–1335.
- Simon, W. (2009). Follow-up psychotherapy outcome of patients with dependent, avoidant and obsessive-compulsive personality disorders: A meta-analytic review. *International Journal of Psychiatry in Clinical Practice*, 13, 153–165.
- Slavich, G. M., & Irwin, M. R. (2014). From stress to inflammation and major depressive disorder: A social signal transduction theory of depression. *Psychological Bulletin*, 140, 774–815.
- Slotema, C. W., Blom, J. D., Hoek, H. W., & Sommer, I. E. (2010). Should we expand the toolbox of psychiatric treatment methods to include repetitive transcranial magnetic stimulation (rTMS)? A meta-analysis of the efficacy of rTMS in psychiatric disorders. *Journal of Clinical Psychiatry*, 71, 873–884.
- Snyder, S. H. (1996). *Drugs and the brain*. New York: Freeman.
- Sobczak, S., Honig, A., Nicolson, N. A., & Riedel, W. J. (2002). Effects of acute tryptophan depletion on mood and cortisol release in first-degree relatives of type I and type II bipolar patients and healthy matched controls. *Neuropsychopharmacology*, 27, 834–842.
- Solomon, D. A., Keller, M. B., Leon, A. C., Mueller, T. I., Lavori, P. W., Shea, M. T., et al. (2000). Multiple recurrences of major depressive disorder. *American Journal of Psychiatry*, 157, 229–233.
- Spek, V., Cuijpers, P., Nyklicek, I., Riper, H., Keyzer, J., & Pop, V. (2007). Internet-based cognitive behaviour therapy for symptoms of depression and anxiety: A meta-analysis. *Psychological Medicine*, 37, 319–328.

- Stack, S. (2000). Media impacts on suicide: A quantitative review of 293 findings. *Social Science Quarterly*, 81, 957–971.
- Styron, W. (1992). *Darkness visible: A memoir of madness*. New York: Vintage.
- Sullivan, P. F., Daly, M. J., & O'Donovan, M. (2012). Genetic architectures of psychiatric disorders: The emerging picture and its implications. *Nature Reviews Genetics*, 13(8), 537–551.
- Sullivan, P. F., Neale, M. C., & Kendler, K. S. (2000). Genetic epidemiology of major depression: Review and metaanalysis. *American Journal of Psychiatry*, 157, 1552–1562.
- Szasz, T. S. (1999). *Fatal freedom: The ethics and politics of suicide*. Westport, CT: Praeger.
- Tarrier, N., Taylor, K., & Gooding, P. (2008). Cognitive-behavioral interventions to reduce suicide behavior. *Behavior Modification*, 32, 77–108.
- Thase, M. E., & Rush, A. J. (1997). When at first you don't succeed: Sequential strategies for antidepressant nonresponders. *Journal of Clinical Psychiatry*, 58, 23–29.
- Tondo, L., Isacson, G., & Baldessarini, R. (2003). Suicidal behaviour in bipolar disorder: risk and prevention. *CNS Drugs*, 17, 491–511.
- Todd, N. J., Jones, S. H., Hart, A., & Lobban, F. A. (2014). A web-based self-management intervention for Bipolar Disorder 'living with bipolar': a feasibility randomised controlled trial. *Journal of Affective Disorders*, 169, 21–29.
- Treadway, M. T., & Zald, D. H. (2011). Reconsidering anhedonia in depression: Lessons from translation neuroscience. *Neuroscience and Biobehavioral Reviews*, 35, 537–555.
- Treynor, W., Gonzalez, R., & Nolen-Hoeksema, S. (2003). Rumination reconsidered: A psychometric analysis. *Cognitive Therapy and Research*, 27, 247–259.
- Trivedi, M. H., Rush, A. J., Wisniewski, S. R., Nierenberg, A. A., Warden, D., Ritz, L., et al. (2006). Evaluation of outcomes with citalopram for depression using measurement-based care in STAR\*D: Implications for clinical practice. *American Journal of Psychiatry*, 163, 28–40.
- Turner, E. H., Matthews, A. M., Linardatos, E., Tell, R. A., & Rosenthal, R. (2008). Selective publication of antidepressant trials and its influence on apparent efficacy. *New England Journal of Medicine*, 358, 252–260.
- Uher, R., & McGuffin, P. (2010). The moderation by the serotonin transporter gene of environmental adversity in the etiology of depression: 2009 update. *Molecular Psychiatry*, 15, 18–22.
- UK ECT Review Group. (2003). Efficacy and safety of electro-convulsive therapy in depressive disorders: A systematic review and meta-analysis. *The Lancet*, 361, 799–808.
- van den Biggelaar, A. H., Gussekloo, J., de Craen, A. J., Frolich, M., Stek, M. L., van der Mast, R. C., et al. (2007). Inflammation and interleukin-1 signaling network contribute to depressive symptoms but not cognitive decline in old age. *Experimental Gerontology*, 42, 693–701.
- van der Sande, R., Buskens, E., Allart, E., van der Graaf, Y., & van Engeland, H. (1997). Psychosocial intervention following suicide attempt: A systematic review of treatment interventions. *Acta Psychiatrica Scandinavica*, 96, 43–50.
- van Orden, K. A., Cukrowicz, K. C., Witte, T. K., Braithwaite, S. R., & Joiner, T. E. (2010). The interpersonal theory of suicide. *Psychological Review*, 117, 575–600.
- van Orden, K. A., Witte, T. K., Gordon, K. H., Bender, T. W., & Joiner, T. E. (2008). Suicidal desire and the capability for suicide: Tests of the interpersonal-psychological theory of suicidal behavior among adults. *Journal of Consulting and Clinical Psychology*, 76, 72–83.
- Videbech, P., & Ravnkilde, B. (2004). Hippocampal volume and depression: A meta-analysis of MRI studies. *American Journal of Psychiatry*, 161, 1957–1966.
- Vieta, E., Martinez-De-Osaba, M. J., Colom, F., Martinez-Aran, A., Benabarre, A., & Gasto, C. (1999). Enhanced corticotropin response to corticotropin-releasing hormone as a predictor of mania in euthymic bipolar patients. *Psychological Medicine*, 29, 971–978.
- Vittengl, J. R., Clark, L. A., Dunn, T. W., & Jarrett, R. B. (2007). Reducing relapse and recurrence in unipolar depression: A comparative meta-analysis of cognitive-behavior therapy's effects. *Journal of Consulting and Clinical Psychology*, 75, 475–488.
- Wakefield, J. C. (2011). Should uncomplicated bereavement-related depression be reclassified as a disorder in the DSM-5? Response to Kenneth S. Kendler's statement defending the proposal to eliminate the bereavement exclusion. *Journal of Nervous and Mental Disease*, 199, 203–208.
- Watkins, E. R. (2008). Constructive and unconstructive repetitive thought. *Psychological Bulletin*, 134, 163–206.
- Watson, D. (2005). Rethinking the mood and anxiety disorders: A qualitative hierarchical model for DSM-V. *Journal of Abnormal Psychology*, 114, 522–536.
- Watson, D. (2009). Differentiating the mood and anxiety disorders: A quadripartite model. *Annual Review of Clinical Psychology*, 5, 221–247.
- Watson, D., O'Hara, M. W., & Stuart, S. (2008). Hierarchical structures of affect and psychopathology and their implications for the classification of emotional disorders. *Depression and Anxiety*, 25, 282–288.
- Watson, S., Thompson, J. M., Ritchie, J. C., Ferrier, I. N., & Young, A. H. (2006). Neuropsychological impairment in bipolar disorder: The relationship with glucocorticoid receptor function. *Bipolar Disorders*, 8, 85–90.
- Wehr, T. A., Duncan, W. C., Sher, L., Aeschbach, D., Schwartz, P. J., Turner, E. H., et al. (2001). A circadian signal of change of season in patients with seasonal affective disorder. *Archives of General Psychiatry*, 58, 1108–1114.
- Weiner, B., Frieze, L., Kukla, A., Reed, L., Rest, S., & Rosenbaum, R. M. (1971). *Perceiving the causes of success and failure*. New York: General Learning Press.

- Weisberg, R. W. (1994). Genius and madness? A quasi-experimental test of the hypothesis that manic-depression increases creativity. *Psychological Science*, 5, 361–367.
- Wells, J. E. (2006). Twelve-month prevalence. In M. A. Oakley Browne, J. E. Wells, & K. M. Scott (Eds.), *Te Rau Hinengaro: The New Zealand Mental Health Survey*. Wellington: Ministry of Health.
- Wender, P. H., Kety, S. S., Rosenthal, D., Schulsinger, F., Ortmann, J., & Lunde, I. (1986). Psychiatric disorders in the biological and adoptive families of adopted individuals with affective disorders. *Archives of General Psychiatry*, 43, 923–929.
- Whisman, M. A., & Bruce, M. L. (1999). Marital dissatisfaction and incidence of major depressive episode in a community sample. *Journal of Abnormal Psychology*, 108, 674–678.
- Winchel, R. M., Stanley, B., & Stanley, M. (1990). Biochemical aspects of suicide. In S. J. Blumenthal & D. J. Kupfer (Eds.), *Suicide over the life cycle: Risk factors, assessment and treatment of suicidal patterns* (pp. 97–126). Washington, DC: American Psychiatric Press.
- Wisner, K. L., Sit, D. K., McShea, M. C., Rizzo, D. M., Zoretich, R. A., Hughes, C. L., et al. (2013). Onset timing, thoughts of self-harm, and diagnoses in postpartum women with screen-positive depression findings. *Journal of the American Medical Association Psychiatry*, 70, 490–498.
- Young, M. A., Watel, L. G., Lahmeyer, H. W., & Eastman, C. I. (1991). The temporal onset of individual symptoms in winter depression: Differentiating underlying mechanisms. *Journal of Affective Disorders*, 22, 191–197.

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## CHAPTER 4

# Anxiety, obsessive-compulsive and trauma-related disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 4.1** describe the clinical features of the anxiety disorders
  - 4.2** describe how the anxiety disorders tend to co-occur and understand how gender and culture influence the prevalence of anxiety disorders
  - 4.3** recognise commonalities in aetiology across the anxiety disorders, as well as the factors that shape the expression of specific anxiety disorders
  - 4.4** describe treatment approaches that are common across the anxiety disorders and how treatment approaches are modified for the specific anxiety disorders
  - 4.5** define the symptoms and epidemiology of the obsessive-compulsive and related disorders and the trauma-related disorders
  - 4.6** describe the commonalities in the aetiology of obsessive-compulsive and related disorders, as well as the factors that shape the expression of the specific disorders within this cluster
  - 4.7** describe the medication and psychological treatments for the obsessive-compulsive and related disorders
  - 4.8** summarise how the nature and severity of the trauma, as well as biological and psychological risk factors, contribute to whether trauma-related disorders develop
  - 4.9** describe the medication and psychological treatments for the trauma-related disorders.
-

## OPENING SCENARIO

Jenny was a 23-year-old student completing her first year of a medical degree. The year had been a hard one, not only because of the long hours and academic challenges of study but also because her mother had developed cancer. One day, while attending rounds, Jenny found herself feeling lightheaded and dizzy. During rounds, the attending physician would ask students to diagnose and explain a given case and on that day Jenny became extremely worried about whether she would be able to answer these questions when her turn came. As she thought about all this, her heart began to pound and her palms began to sweat. Overwhelmed by a deep sense of fear that something was horribly wrong, she abruptly fled the room without explaining her departure.

Later in the day, she wanted to explain leaving rounds but could not think of a good way to describe the situation to the attending physician. That night, she could not sleep, wondering what had happened and worrying about whether it would happen again. She worried about how this would affect her ability not only to take part in rounds but also to perform well in other roles, such as leading a small research group and meeting with other medical staff and clients. One week later, while driving to university, she experienced a sudden attack of similar symptoms, which forced her to pull off to the side of the road. She took the day off. Over the next several weeks, she began to avoid public situations as much as possible because she feared being humiliated by the return of these symptoms. She avoided study groups and going out with friends and she turned down opportunities for training that involved public interviews of patients. She resigned from the choir that she had enjoyed being a part of for several years. Despite her withdrawal, she experienced three more attacks, each in unexpected situations. She began to think that maybe a medical degree was a poor choice for her because she had such deep fears about experiencing another attack during rounds. After she read about panic disorder in one of her textbooks, she decided to visit a psychologist. The psychologist confirmed that she was experiencing panic disorder and they started cognitive-behavioural treatment.

### QUESTIONS

1. What might be some techniques Jenny could use to help alleviate her symptoms?
2. Now that she is receiving psychological care, what might some of the next steps be to ensure Jenny maintains her lifestyle?

## Introduction

Very few of us go through even a week of our lives without experiencing anxiety or fear. In the first part of this chapter, we focus on a group of disorders called **anxiety disorders**.

Both anxiety and fear play a significant role in these disorders. **Anxiety** is defined as apprehension over an anticipated problem. In contrast, **fear** is defined as a reaction to immediate danger. Psychologists focus on the ‘immediate’ aspect of fear (a *present* threat) versus the ‘anticipated’ aspect of anxiety (a *future* threat). Thus, a person facing a snake experiences fear, whereas a university student concerned about the possibility of unemployment after graduation experiences anxiety.

Both anxiety and fear can involve arousal or sympathetic nervous system activity. Anxiety often involves moderate arousal and fear involves higher arousal. At the low end, a person experiencing anxiety may feel no more than restless energy and physiological tension; at the high end, a person experiencing fear may sweat profusely, breathe rapidly and feel an overpowering urge to run.

Anxiety and fear are not necessarily ‘bad’; in fact, both are adaptive. Fear is fundamental for ‘fight-or-flight’ reactions — it triggers rapid changes in the sympathetic nervous system to prepare the body for escape or fighting. In the right circumstance, fear saves lives. In some anxiety disorders, though, the fear system seems to misfire — a person experiences fear at a time when there is no danger in the environment. This is most vividly seen in panic attacks, which we will discuss later in this chapter.

Anxiety is adaptive in helping us notice and plan for future threats — that is, to increase our preparedness, to help people avoid potentially dangerous situations and to think through potential problems before they happen. In numerous laboratory studies conducted over the past century, a small degree of anxiety has been found to improve performance on laboratory tasks (Yerkes & Dodson, 1908). Ask anyone with extreme test anxiety, though, and they will tell you that too much anxiety interferes with performance. Anxiety, then, provides a classic example of an inverse U-shaped curve with performance — an absence of anxiety is a problem, a little anxiety is adaptive and a lot of anxiety is detrimental.

In this section, we examine the major anxiety disorders included in DSM-5: specific phobias, social anxiety disorder, panic disorder, agoraphobia and generalised anxiety disorder. Later in the chapter, obsessive-compulsive disorder and trauma-related disorders are explored. These have a good deal in common with the anxiety disorders but are also distinct in some important ways. All of the disorders covered in this chapter involve excessive amounts of anxiety and, with the exception of generalised anxiety disorder, all involve tendencies to experience unusually intense fear (Cox, Clara, & Enns, 2002).

Anxiety disorders as a group are the most common type of psychological disorder. In a 2007 survey, over 2 million Australians (14 percent of the population) reported experiencing anxiety within a 12-month period (ABS, 2008). Phobias are particularly common. As a group, anxiety disorders are very costly to society and to people with the disorders. These disorders are associated with twice the average rate of medical costs (Simon, Ormel, VonKroff, & Barlow, 1995), higher risk of cardiovascular disease and other medical conditions (Roest, Martens, de Jonge, & Denollet, 2010; Stein et al., 2014), twice the risk of suicidal ideation and attempts (Sareen et al., 2005), high rates of unemployment and days out of work (Alonso et al., 2011), and high rates of marital discord (Whisman & Bruce, 1999) compared to people without a psychological diagnosis. All of the anxiety disorders are associated with substantial decrements in the quality of life (Olatunji, Cisler, & Tolin, 2007). The following quote illustrates some of the ways that anxiety can influence daily life.

On ordinary days, doing ordinary things — reading a book, lying in bed, talking on the phone, sitting in a meeting, playing tennis — I have thousands of times been stricken by a pervasive sense of existential dread and been beset by nausea, vertigo, shaking and a panoply of other physical symptoms. In these instances, I have sometimes been convinced that death or something somehow worse, was imminent. (Stossel, 2014, p. 2)

We begin by defining the symptoms of the anxiety disorders before turning to their aetiology. Like most disorders, many different paradigms have helped shed light on the anxiety disorders. Hence, throughout our discussions of aetiology, we look at issues from various perspectives, with particular focus on genetic, neurobiological, personality, cognitive and behavioural research. Finally, we consider the treatment of the anxiety disorders.

## 4.1 Clinical descriptions of the anxiety disorders

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**LEARNING OUTCOME 4.1** Describe the clinical features of the anxiety disorders.

There is a lot of overlap in the way the various anxiety disorders are defined. For each disorder, several criteria must be met for a DSM-5 diagnosis to be made.

- Symptoms interfere with important areas of functioning or cause marked distress.
- Symptoms are not caused by a drug or a medical condition.
- Symptoms persist for at least six months or at least one month for panic disorder.
- The fears and anxieties are distinct from the symptoms of another anxiety disorder.

Each disorder, though, is defined by a different set of symptoms related to anxiety or fear (see table 4.1 for a brief summary).

**TABLE 4.1** Overview of the major DSM-5 anxiety disorders

Disorder	Description
Specific phobia	Fear of objects or situations that is out of proportion to any real danger
Social anxiety disorder	Fear of unfamiliar people or social scrutiny
Panic disorder	Anxiety about recurrent panic attacks
Agoraphobia	Anxiety about being in places where escaping or getting help would be difficult if anxiety symptoms occurred
Generalised anxiety disorder	Uncontrollable worry

## Specific phobias

A **specific phobia** is a disproportionate fear caused by a specific object or situation, such as fear of flying, fear of snakes or fear of heights. The person recognises that the fear is excessive but still goes to great lengths to avoid the feared object or situation. In addition to fear, the object of a phobia may elicit intense disgust (Olatunji, Etzel, Tomarken, Ciesielski, & Deacon, 2011). The names for these fears consist of a Greek word for the feared object or situation followed by the suffix *-phobia* (derived from the name of the Greek god Phobos, who frightened his enemies). Two of the more common phobias are claustrophobia (fear of closed spaces) and acrophobia (fear of heights). Specific phobias tend to cluster around a small number of feared objects and situations (see table 4.2). A person with a specific phobia for one type of object or situation is very likely to have a specific phobia for a second object or situation — that is, specific phobias are highly comorbid (Kendler, Myers, Prescott, & Neale, 2001). The clinical case of Jan provides a glimpse of how specific phobias can interfere with important life goals.

### DSM-5

#### DSM-5 criteria for specific phobia

- A phobia is a marked and disproportionate fear consistently triggered by specific objects or situations.
- The object or situation is avoided or else endured with intense anxiety.

**TABLE 4.2** Types of specific phobias

Type of phobia	Examples of the feared object	Associated characteristics
Animal	Snakes, insects	Generally begins during childhood
Natural environment	Storms, heights, water	Generally begins during childhood
Blood, injection, injury	Blood, injury, injections or other invasive medical procedures	Clearly runs in families; profile of heart rate slowing and possible fainting when facing feared stimulus (LeBeau et al., 2010)
Situational	Public transportation, tunnels, bridges, elevators, flying, driving, closed spaces	Tends to begin either in childhood or in mid-20s
Other	Choking, contracting an illness, etc.; children's fears of loud sounds, clowns, etc.	

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Acrophobia, or phobia of heights, is common. People with this fear would find a ferris wheel terrifying. Other specific phobias include fears of animals, injections and enclosed spaces.



#### CLINICAL CASE

##### Jan

Jan, a 42-year-old woman, had been offered a high-paying job in Cairns. She was considering declining the offer because it would force her to live in an area known for having snakes. Before making this decision, she decided to see a therapist. During her first meeting with the therapist, she described a litany of ways she had avoided any contact with anything remotely resembling a snake. She had steered clear of outdoor activities, TV programs on nature and even her children's books on nature. Although she had been able to cope with her fears without too many negative consequences so far, the idea of living in an area with snakes had greatly increased her apprehension. Aside from her phobia, Jan reported that she had always been a bit of a nervous person, a trait she shared with her mother.

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##### QUESTIONS

1. What questions might the therapist ask Jan?
2. What might be some ways to help Jan with her phobia?

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One form of specific phobia is an intense fear of blood, injection or injuries.



## Social anxiety disorder

The core feature of **social anxiety disorder** is a persistent, unrealistically intense fear of social situations that might involve being scrutinised by, or even just exposed to, unfamiliar people. Although this disorder is labelled **social phobia** in the DSM-IV-TR, the term *social anxiety disorder* is used in the DSM-5 because the problems caused by it tend to be much more pervasive and to interfere much more with normal activities than the problems caused by other phobias (Liebowitz, Heimberg, Fresco, Travers, & Stein, 2000). In interpersonal interactions, people with social anxiety disorder feel as though 'all eyes are watching them', with others waiting to evaluate them and record any embarrassing acts. Although this may sound like shyness, people with social anxiety disorder avoid more social situations, feel more social discomfort and experience these symptoms for longer periods of their lifetime than do people who are shy (Turner, Beidel, & Townsley, 1990). Many fear that their anxiety will become apparent to others. The most common fears include public speaking, speaking up in meetings or classes, meeting new people and talking to people in authority (Ruscio et al., 2008). Forced to engage in one of these feared activities, the person may spend days in advance thinking about all the possible ways things could go wrong and then spend days after the event reliving the small moments that did go badly with a sense of horror.

The manifestations and outcomes of social anxiety disorder vary greatly. These can range in severity from a few specific fears to a generalised host of fears. For example, some people might be anxious about speaking in public but not about other social situations. In contrast, others report fearing most social situations. Those with a broader array of fears are likely to experience more comorbid depression and alcohol abuse (Acarturk, de Graaf, van Straten, Have, & Cuijpers, 2008). Although many who suffer with social anxiety are withdrawn and submissive, people vary in how they cope with the threat of social rejection and a small proportion respond with overtly hostile and aggressive behaviour (Kashdan & McKnight, 2010). People with social anxiety disorder often work in occupations far below their talents because of their extreme social fears. Many would rather work in an unrewarding job with limited social demand than deal with social situations every day.

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Social anxiety disorder typically begins in adolescence and interferes with developing friendships.



#### DSM-5

##### DSM-5 criteria for social anxiety disorder

- People with social anxiety disorder have a marked and disproportionate fear consistently triggered by exposure to potential social scrutiny.
- Exposure to trigger situations leads to intense anxiety about being evaluated negatively — these situations are avoided or else endured with intense anxiety.

#### CLINICAL CASE

##### Maureen

Maureen, a 30-year-old accountant, sought psychotherapy after reading a newspaper notice advertising group therapy for people with difficulties in social situations. Maureen appeared nervous during the interview and described feeling intensely anxious in conversations with others. She described the problem as becoming worse over the years, to the point where she no longer interacted socially with anyone other than her husband. She would not even go to the supermarket for fear of having to interact with people. Maureen deeply feared being perceived as stupid. This fear made her so nervous that she would often stammer or forget what she was going to say while talking to others, thus adding to her apprehension that others would see her as stupid, and creating a vicious circle of ever-increasing fear.

##### QUESTIONS

1. What are the difficulties in the treatment of Maureen?
2. Do you think Maureen is at higher risk of developing other psychological disorders?

Among people with social anxiety disorder, at least a third also meet the criteria for a diagnosis of avoidant personality disorder (see the chapter on personality and personality disorders; Chavira, Stein, & Malcarne, 2002). The symptoms of the two conditions overlap a great deal, as does the genetic vulnerability for the two conditions (Reichborn-Kjennerud et al., 2007). Avoidant personality disorder, though, is a more severe disorder with an earlier onset and more pervasive symptoms.

Social anxiety disorder generally begins during adolescence, when peer relationships become particularly important. For some, however, the symptoms first emerge during childhood. Without treatment, social anxiety disorder tends to be chronic (Yonkers, Bruce, Dyck, & Keller, 2003).

## Panic disorder

**Panic disorder** is characterised by recurrent panic attacks that are unrelated to specific situations and by worry about having more panic attacks (see the opening scenario at the beginning of this chapter). A **panic attack** is a sudden attack of intense apprehension, terror and feelings of impending doom, accompanied by at least four other symptoms. Physical symptoms can include shortness of breath, heart palpitations, nausea, upset stomach, chest pain, feelings of choking and smothering, dizziness, light-headedness, faintness, sweating, chills, heat sensations, numbness or tingling sensations, and trembling. Other symptoms include **depersonalisation** (a feeling of being outside one's body); **derealisation** (a feeling of the world not being real); and fears of losing control, of going crazy or even of dying. Not surprisingly, people often report that they have an intense urge to flee whatever situation they are in when a panic attack occurs. The symptoms tend to come on very rapidly and reach a peak of intensity within 10 minutes. Many people seek emergency medical care when they first experience a panic attack because they are terrified that they are having a heart attack.

### DSM-5

#### DSM-5 criteria for panic disorder

- People with panic disorder experience recurrent unexpected panic attacks.
- Panic disorder can be diagnosed if the person experiences at least one month of concern or worry about the possibility of more attacks occurring or the consequences of an attack, or maladaptive behavioural changes because of the attacks.

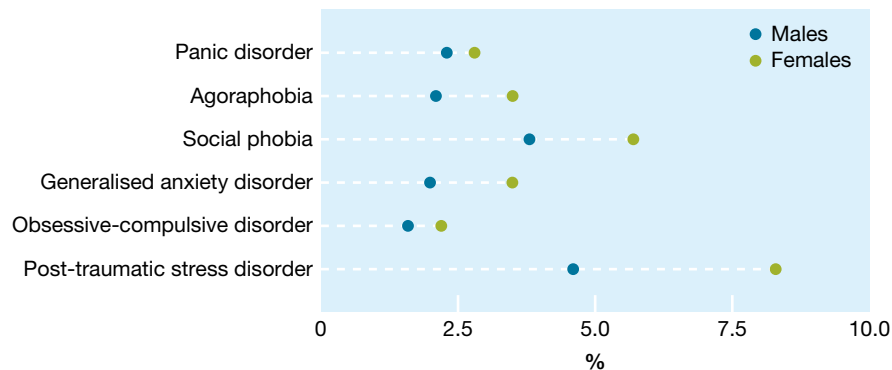
As noted above, we can think about a panic attack as a misfire of the fear system: physiologically, the person experiences a level of sympathetic nervous system arousal matching what most people might experience when faced with an immediate threat to life. Because the symptoms are inexplicable, the person tries to make sense of the experience. A person who begins to think that he or she is dying, losing control or going crazy is likely to feel even more fear. Among people with panic disorder, 90 percent report just these types of beliefs when panic attacks occur.

The diagnostic criteria for panic disorder require more than the presence of recurrent panic attacks. According to the DSM criteria, a person must experience recurrent panic attacks that are unexpected. Panic attacks that are triggered by specific situations, such as seeing a snake, are typically related to a phobia and should not be considered in diagnosing panic disorder. Beyond the occurrence of unexpected panic attacks, DSM criteria also specify that the person must worry about the attacks or change his or her behaviour because of the attacks for at least one month. Hence, in making this diagnosis, the response to panic attacks is as important as the attacks themselves.

Remember that the criteria for panic disorder specify that panic attacks must be recurrent. It is fairly common for people to experience a single panic attack — more than 40 percent of people in Australia will experience at least one panic attack during their lifetime (ABS, 2008, cited in beyondblue, n.d.). As figure 4.1 shows, though, many fewer people develop full-blown panic disorder. Among those who develop panic disorder, the onset is typically in adolescence. The symptoms of panic disorder tend to

wax and wane over time (Nay, Brown, & Roberson-Nay, 2013). It can take a heavy toll; for example, as many as one-quarter of people with panic disorder report being unemployed for more than five years (Leon, Portera, & Weissman, 1995).

**FIGURE 4.1** Twelve-month anxiety disorders; persons who met criteria for diagnosis of a lifetime mental disorder (with hierarchy) and had symptoms in the 12 months prior to interview. A person may have had more than one anxiety disorder.



Source: ABS (2008).

People with panic disorder often seek cardiac tests because they are frightened by changes in their heart rate.



## Agoraphobia

**Agoraphobia** (from the Greek *agora*, meaning ‘marketplace’) is defined by anxiety about situations in which it would be embarrassing or difficult to escape if anxiety symptoms occurred, such as being in a crowded place like a shopping centre or church. Sometimes the situations are those that are difficult to escape from, such as trains, bridges or long road trips. As a consequence of their fear of these situations, many people with agoraphobia are virtually unable to leave their house and even those who can leave do so only with great distress.

In the DSM-IV-TR, agoraphobia is coded as a subtype of panic disorder, but the DSM-5 includes agoraphobia as a separate diagnosis. This change brings the DSM in line with the International Classification of Diseases (ICD) diagnostic system, which has long recognised agoraphobia as a separate diagnosis. The new diagnosis also fits evidence from research studies. Indeed, five different large epidemiological studies suggest that at least half of people with agoraphobia symptoms do not experience panic attacks (Andrews, Charney, Sirovatka, & Regier, 2009). Those who do not experience panic attacks are concerned about what will happen if other anxiety symptoms develop. The new diagnosis also fits with evidence that agoraphobia is related to significant impairment in daily functioning. The effects of agoraphobia on quality of life are as severe as those observed for the other anxiety disorders (Wittchen, Gloster, Beesdo-Baum, Fava, & Craske, 2010).

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People with agoraphobia often find crowds very distressing because escape would be difficult if anxiety symptoms occurred.



## DSM-5

### DSM-5 criteria for agoraphobia

- People who meet the criteria for agoraphobia experience a disproportionate and marked fear or anxiety about at least two situations where it would be difficult to escape or receive help in the event of incapacitation, embarrassing symptoms or panic-like symptoms, such as being outside of the home alone; travelling on public transportation; being in open spaces such as parking lots and marketplaces; being in enclosed spaces such as shops, theatres or cinemas; or standing in line or being in a crowd.
- These situations consistently provoke fear or anxiety.
- These situations are avoided, require the presence of a companion, or are endured with intense fear or anxiety.

## Generalised anxiety disorder

The central feature of **generalised anxiety disorder (GAD)** is worry. Like Joe in the clinical case below, people with GAD are persistently worried, often about minor things. The term *worry* refers to the cognitive tendency to chew on a problem and to be unable to let go of it (Mennin, Heimberg, & Turk, 2004). Often, worry continues because a person cannot settle on a solution to the problem. Most of us worry from time to time, but the worries of people with GAD are excessive, uncontrollable and long-lasting. The worries of people with GAD centre on the same types of threats that worry most of us: relationships, health, finances and daily hassles (Roemer, Molina, & Borkovec, 1997) — but they worry more about these issues, as illustrated by the following passage:

I had graduated from college the year before, with honors. I had a prestigious job, loyal friends, a good apartment I shared with a bright and beautiful girlfriend, and as much money as I needed. Yet every day was torture. I slept fitfully, with recurring nightmares — tsunamis, feral animals, the violent deaths of loved ones. I had intestinal cramps and nausea and headaches. A sense of impending catastrophe colored every working moment. Worse, I had the distinct sense that catastrophe had already occurred. I had made the wrong decisions, gone down the wrong path, screwed up in a ruinous, irrevocable, epoch-making way. (Smith, 2012, pp. 3–4)

Beyond the uncontrollable worries, other symptoms of GAD include difficulty concentrating, tiring easily, restlessness, irritability and muscle tension. GAD is not diagnosed if a person worries only about concerns driven by another psychological disorder; for example, a person with claustrophobia who only worries about being in closed spaces would not meet the criteria for GAD.

## DSM-5

### DSM-5 criteria for generalised anxiety disorder

- People who meet the criteria for generalised anxiety disorder experience excessive anxiety and worry at least 50 percent of days about a number of events or activities (e.g., family, health, finances, work and school).
- The person finds it hard to control the worry.
- The anxiety and worry are associated with at least three (or one in children) of the following:
  - restlessness or feeling keyed up or on edge
  - easily fatigued
  - difficulty concentrating or mind going blank
  - irritability
  - muscle tension
  - sleep disturbance.

GAD typically begins in adolescence, though many people who have GAD report having had a tendency to worry all their lives (Barlow, Blanchard, Vermilyea, Vermilyea, & DiNardo, 1986). Once it develops, GAD is often chronic; in one study, about half of people with GAD reported ongoing symptoms eight years after an initial interview (Yonkers et al., 2003). Perhaps because of the chronicity, GAD is more strongly related to marital dissatisfaction than any other anxiety disorder (Whisman, Sheldon, & Goering, 2000) and people diagnosed with GAD report having few friendships compared to others (Whisman et al., 2000).

#### CLINICAL CASE

##### Joe

Joe, a 24-year-old mechanic, was referred for psychotherapy by his physician, whom he had consulted because of difficulty falling asleep. He was visibly distressed during the entire initial interview, with a furrowed brow and continuous fidgeting. Although he first described worries about his health, a picture of pervasive anxiety soon emerged. He reported that he nearly always felt tense and he seemed to worry about everything. He was apprehensive of disasters that could befall him as he interacted with other people and worked, and he described worrying much of the time about his finances, his inability to establish a romantic relationship and other issues. He reported a long history of difficulties relating to others, which had led to his being fired from several jobs. As he put it, 'I really like people and try to get along with them, but I fly off the handle too easily. Little things upset me too much.' Joe reported that he had always felt more nervous than other people but that his anxiety had become much worse after a romantic breakup one year ago.

##### QUESTIONS

1. Have you encountered people like Joe before? If so, how was your relationship with them?
2. What DSM-5 criteria for generalised anxiety disorder does Joe demonstrate?

## 4.2 Comorbidity, gender and culture in anxiety disorders

**LEARNING OUTCOME 4.2** Describe how the anxiety disorders tend to co-occur and understand how gender and culture influence the prevalence of anxiety disorders.

### Comorbidity in anxiety disorders

More than half of people with one anxiety disorder meet the criteria for another anxiety disorder during their lifetime (Brown, Campbell, Lehman, Grisham, & Mancill, 2001). Anxiety disorders are also highly comorbid with other disorders. Three-quarters of people with an anxiety disorder meet the diagnostic criteria for at least one other psychological disorder (Kessler et al., 1997). More specifically, about 60 percent of people in treatment for anxiety disorders meet the diagnostic criteria for major depression (Brown et al., 2001). We discuss this overlap in focus on discovery 3.4. Other conditions commonly comorbid with anxiety disorders include substance abuse (Jacobsen, Southwick, & Kosten, 2001) and personality disorders (Johnson, Weissman, & Klerman, 1992). As with many disorders, comorbidity is associated with greater severity and poorer outcomes of the anxiety disorders (Newman, Moffitt, Caspi, & Silva, 1998; Newman, Schmitt, & Voss, 1997).

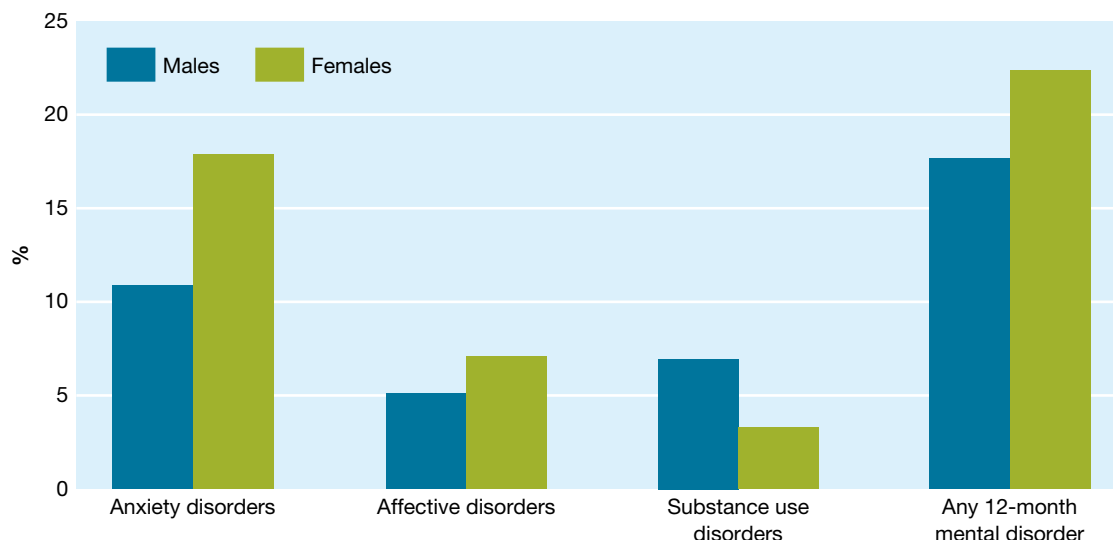
### Gender and cultural factors in anxiety disorders

It is well known that gender and culture are closely tied to the risk for anxiety disorders and to the specific types of symptoms that a person develops. As you will see, however, there are still some puzzles about why these patterns exist.

## Gender

Women are more vulnerable to anxiety disorders than men, at 18 percent compared with 11 percent for their male counterparts (ABS, 2008). Different theories have been proposed to explain why women are more likely to develop anxiety disorders than men. First, women may be more likely to report their symptoms. Social factors, such as gender roles, are also likely to play a role. For example, men may experience more social pressure than women to face fears — as you will see below, facing fears is the basis for one of the most effective treatments available. Women may also experience different life circumstances than do men. For example, women are much more likely than men to be sexually assaulted during childhood and adulthood (Tolin & Foa, 2006). These traumatic events may interfere with developing a sense of control over one's environment and having less control over one's environment may set the stage for anxiety disorders. Men may be raised to believe more in their personal control over situations as well. It also appears that women show more biological reactivity to stress than do men (Olff, Langeland, Draijer, & Gersons, 2007), perhaps as a result of these cultural and psychological influences. Although the gender gap is not fully understood, it is an important phenomenon.

**FIGURE 4.2** Twelve-month mental disorders, by major disorder group; persons who met criteria for diagnosis of a lifetime mental disorder (with hierarchy) and had symptoms in the 12 months prior to interview. A person may have had more than one mental disorder.



Source: ABS (2008).

## Culture

People in every culture seem to experience problems with anxiety disorders, but culture and environment influence what people come to fear (Kirmayer, 2001). ‘If you live near a volcano you’re going to fear lava. If you live in the rainforest you’re going to fear malaria’ (Smith, 2012, p. 72). *Kayak-angst*, a disorder that is similar to panic disorder, occurs among the Inuit people of western Greenland; seal hunters who are alone at sea may experience intense fear, disorientation and concerns about drowning.

Several other culturally relevant syndromes provide examples of how culture and environment may shape the expression of an anxiety disorder. For example, in Japan a syndrome called *taijin kyofusho* involves fear of displeasing or embarrassing others; people with this syndrome typically fear such things as making direct eye contact, blushing, having body odour or having a bodily deformity. The symptoms of this disorder overlap with those of social anxiety disorder, but the focus on the feelings of others is distinct. Perhaps this focus is related to characteristics of traditional Japanese culture that encourage deep concern for the feelings of others (McNally, 1997). Other syndromes, such as *koro* (a sudden fear

that one's genitals will recede into the body — reported in southern and eastern Asia), *shenkui* (intense anxiety and somatic symptoms attributed to the loss of semen, as through masturbation or excessive sexual activity — reported in China and similar to other syndromes reported in India and Sri Lanka) and *susto* (fright-illness, the belief that a severe fright has caused the soul to leave the body — reported in Latin America and among Latinos in the United States), also involve symptoms similar to those of the anxiety disorders defined in the DSM. As with the Japanese syndrome *taijin kyofusho*, the objects of anxiety and fear in these syndromes relate to environmental challenges as well as to attitudes that are prevalent in the cultures where the syndromes occur.

Beyond culturally relevant syndromes, the prevalence of anxiety disorders varies across cultures. This is not surprising given that cultures differ with regard to factors such as attitudes towards psychological disorders, stress levels, the nature of family relationships and the prevalence of poverty — all of which are known to play a role in the occurrence or reporting of anxiety disorders. For example, in Taiwan and Japan, the prevalence of anxiety disorders seems to be quite low; however, this may reflect a strong stigma associated with having mental problems, which could lead to under-reporting in those countries (Kawakami, Shimizu, Haratani, Iwata, & Kitamura, 2004). In Cambodia and among Cambodian refugees, very elevated rates of panic disorder (often diagnosed traditionally as *kyol goeu* or 'wind overload') have been reported, perhaps because of the extreme stress experienced by Cambodians over the past several decades (Hinton, Ba, Peou, & Um, 2000; Hinton, Um, & Ba, 2001).

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Disorders similar to panic attacks occur cross-culturally. Among the Inuit, *kayak-angst* is defined by intense fear in lone hunters.



For some time, researchers have thought that people from different cultures express symptoms of psychological distress and anxiety in different ways. For example, many thought that those from some cultures would express their emotional concerns through somatic complaints, but it now seems that this conclusion might reflect sampling problems — that is, researchers often studied anxiety and depression in psychological clinics in the United States but in medical clinics in other cultures. One can imagine that a person seeing a medical doctor might be likely to emphasise somatic concerns! Indeed, many people, regardless of culture, tend to describe anxiety and depression initially in terms of bodily sensations

when visiting a medical clinic. When researchers interview people in similar settings and ask specifically about psychological concerns, the ratio of somatic to psychological symptoms expressed appears to be much more similar across cultures (Kirmayer, 2001).

## 4.3 Aetiology of anxiety disorders

**LEARNING OUTCOME 4.3** Recognise commonalities in aetiology across the anxiety disorders, as well as the factors that shape the expression of specific anxiety disorders.

As we consider risk factors associated with anxiety disorders, we begin by describing a set of factors that seem to increase risk for all of the anxiety disorders. The existence of such risk factors may help explain why people with one anxiety disorder are likely to develop a second one — that is, some risk factors increase the odds of having more than one anxiety disorder (for example, social anxiety disorder and panic disorder). We will then turn to the question of how each of the specific anxiety disorders arises.

### Common risk factors across the anxiety disorders

Unlike the way we have organised most of the other chapters in this text, we have chosen to begin this section with the behavioural model. We do this because classical conditioning of a fear response is at the heart of many anxiety disorders. Many of the other risk factors, including genes, neurobiological correlates, personality traits and cognition, influence how readily a person can be conditioned to develop a new fear response. Taken together, the risk factors combine to create an increased sensitivity to threat (Craske et al., 2009). Table 4.3 summarises the general risk factors for anxiety disorders.

**TABLE 4.3** Factors that increase general risk for anxiety disorders

Behavioural conditioning (classical and operant conditioning)
Genetic vulnerability
Disturbances in the activity in the fear circuit of the brain
Decreased functioning of gamma-aminobutyric acid (GABA) and serotonin; increased norepinephrine activity
Behavioural inhibition
Neuroticism
Cognitive factors, including sustained negative beliefs, perceived lack of control and attention to cues of threat

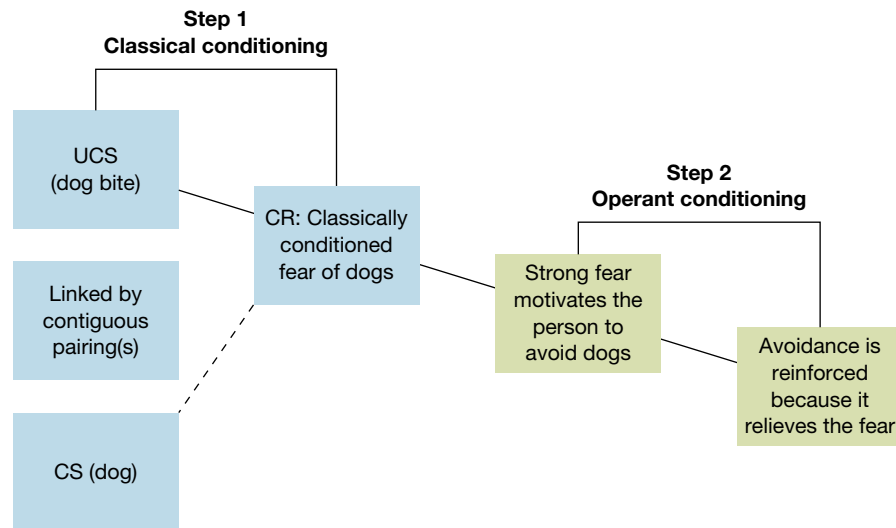
### Fear conditioning

The behavioural theory of anxiety disorders focuses on conditioning. **Mowrer's two-factor model** of anxiety disorders, published in 1947, continues to influence thinking in this area. Mowrer's model suggests two steps in the development of an anxiety disorder (Mowrer, 1947).

1. Through *classical conditioning*, a person learns to fear a neutral stimulus (the conditioned stimulus or CS) that is paired with an intrinsically aversive stimulus (the unconditioned stimulus or UCS).
2. A person gains relief by avoiding the CS. Through *operant conditioning*, this avoidant response is maintained because it is reinforcing (it reduces fear).

Consider the example shown in figure 4.3. Imagine that a man is bitten by a dog and then develops a phobia of dogs. Through classical conditioning, he has learned to associate dogs (the CS) with painful bites (the UCS). This corresponds to step 1 above. In step 2, the man reduces his fear by avoiding dogs as much as possible; the avoidant behaviour is reinforced by the reduction in fear. This second step explains why the phobia isn't extinguished. With repeated exposure to dogs that don't bite, the man would have lost his fear of dogs, but by avoiding dogs, the man gets little or no such exposure.

**FIGURE 4.3** Two-factor model of conditioning as applied to dog phobia



We should note that Mowrer's early version of the two-factor model does not actually fit the evidence very well; several extensions of this model have been developed that fit the evidence better (Mineka & Zinbarg, 1998). One extension considers different ways in which classical conditioning could occur (Rachman, 1977), including:

- direct experience (like the dog bite in the example above)
- modelling (e.g., seeing a dog bite a man or watching a video of a vicious dog attack)
- verbal instruction (e.g., hearing a parent warn that dogs are dangerous).

Beyond considering these different sources of classical conditioning, researchers have shown that people with anxiety disorders seem to acquire fears more readily through classical conditioning and to show a slower extinction of fears once they are acquired (Craske et al., 2009). Most studies of this phenomenon use carefully controlled tests in a laboratory setting. For example, in one study, researchers conditioned people to fear a neutral picture of a Rorschach card (see figure 2.11) by pairing the card with a shock six times (Michael, Blechert, Vriends, Margraf, & Wilhelm, 2007). After receiving six shocks, most participants learned to fear the Rorschach card, as measured by using skin conductance responses to seeing the card — even those without an anxiety disorder developed this conditioned response. Those with and without an anxiety disorder (in this study, panic disorder) differed in the extinction phase of the study, when the card was shown without any shock being provided. People who were not diagnosed with panic disorder showed a quick drop in their fear responses during the extinction phase, but people with panic disorder showed very little decrease in their fear response. Hence, people with panic disorder appear to sustain classically conditioned fears longer. Findings from a meta-analysis of 20 studies suggest that anxiety disorders are related to an elevated propensity to develop fears through classical conditioning and to a slow extinction of those fears once they are acquired (Lissek et al., 2005). Many of the risk factors we describe next could influence this sensitivity to fear conditioning.

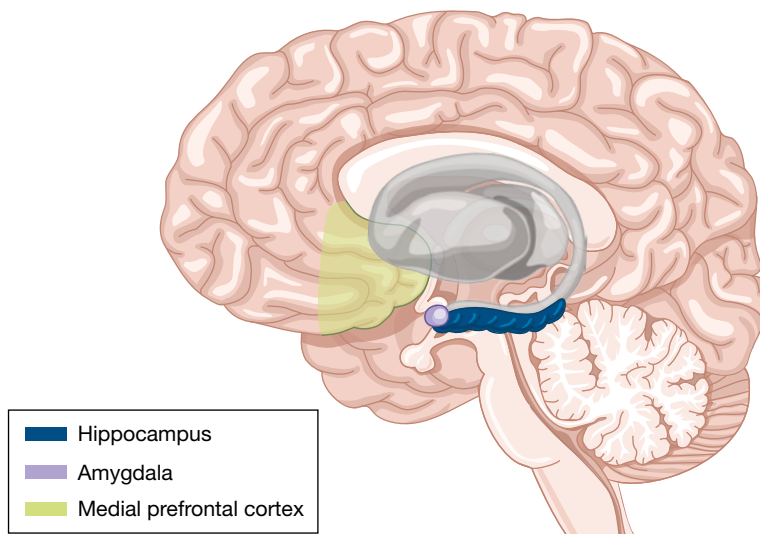
## Genetic factors

Twin studies suggest a heritability of 20–40 percent for specific phobias, social anxiety disorder and GAD, and about 50 percent for panic disorder (Hettema, Neale, & Kendler, 2001; True et al., 1993). Some genes may elevate risk for several different types of anxiety disorder. For example, having a family member with a phobia is related to increased risk of developing not only a phobia but also other anxiety disorders (Kendler et al., 2001). Other genes may elevate risk for a specific type of anxiety disorder (Hettema, Prescott, Myers, Neale, & Kendler, 2005).

## Neurobiological factors: the fear circuit and the activity of neurotransmitters

A set of brain structures called the **fear circuit** (see figure 4.4) is engaged when people feel anxious or fearful (Malizia, 2003). One important part of the fear circuit is the amygdala, a small, almond-shaped structure in the temporal lobe that appears to be involved in assigning emotional significance to stimuli. In animals, the amygdala has been shown to be critical for the conditioning of fear. The amygdala sends signals to a range of different brain structures involved in the fear circuit. Studies suggest that when shown pictures of angry faces (one signal of threat), people with several different anxiety disorders respond with greater activity in the amygdala than do people without anxiety disorders (Blair et al., 2008; Monk et al., 2006). Hence, elevated activity in the amygdala may help explain many different anxiety disorders.

**FIGURE 4.4** Fear and anxiety appear to be related to a set of structures in the brain called the fear circuit. The amygdala and medial prefrontal cortex are particularly involved in anxiety disorders.



The **medial prefrontal cortex** helps to regulate amygdala activity — it is involved in extinguishing fears and also appears to be engaged when people are regulating their emotions (Indovina, Robbins, Nunez-Elizalde, Dunn, & Bishop, 2011; Kim et al., 2011). Researchers have found that adults who meet diagnostic criteria for anxiety disorders display less activity in the medial prefrontal cortex when viewing and appraising threatening stimuli (Britton et al., 2013) and when asked to regulate their emotional responses to threatening stimuli (Goldin, Manber-Ball, Werner, Heimberg, & Gross, 2009). The pathway, or connectivity, linking the amygdala and the medial prefrontal cortex may be deficient among those with anxiety disorders (Kim et al., 2011), which in turn may interfere with the effective regulation and extinction of anxiety (Yehuda & LeDoux, 2007). We will discuss another part of the fear circuit, the locus coeruleus, when we discuss specific anxiety disorders.

## Personality: behavioural inhibition and neuroticism

Some infants show the trait of **behavioural inhibition**, a tendency to become agitated and cry when faced with novel toys, people or other stimuli. This behavioural pattern, which has been described in infants as young as four months old, may be inherited and may set the stage for the later development of anxiety disorders. One study followed infants from 14 months through to 7.5 years; 45 percent of those who showed elevated behavioural inhibition levels at 14 months showed symptoms of anxiety at age 7.5, compared to only 15 percent of those who had shown low behavioural inhibition levels (Kagan & Snidman, 1999). Behavioural inhibition appears to be a particularly strong predictor of social anxiety disorder: infants showing elevated behavioural inhibition were 3.79 times as likely as those with low behavioural inhibition to develop social anxiety disorder by adolescence (Chronis-Tuscano et al., 2009).

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Infants and toddlers showing behavioural inhibition — high anxiety about novel situations and people — are at greater risk of developing anxiety disorders during their lifetime.



Neuroticism is a personality trait defined by the tendency to experience frequent or intense negative affect. In a sample of 7076 adults, neuroticism predicted the onset of both anxiety disorders and depression (de Graaf, Bijl, Ravelli, Smit, & Vollenbergh, 2002). People with high levels of neuroticism were more than twice as likely to develop an anxiety disorder as those with low levels. In another study of 606 adults followed over two years, neuroticism was a major predictor of anxiety and depression (Brown, 2007).

## Cognitive factors

Researchers have focused on several separate cognitive aspects of anxiety disorders. Here, we concentrate on three: sustained negative beliefs about the future, a perceived lack of control and attention to signs of threat.

## **Sustained negative beliefs about the future**

People with anxiety disorders often report believing that bad things are likely to happen. For example, people with panic disorder might believe that they will die when their heart begins to pound, whereas people with social anxiety disorder might believe that they will suffer humiliating rejection if they blush. As pointed out by David Clark and colleagues (Clark et al., 1999), the key issue is not why people think so negatively initially but, rather, how these beliefs are sustained. For example, by the time a person survives 100 panic attacks, you might expect the belief ‘this attack means I am about to die’ would fade. One reason these beliefs might be sustained is that people think and act in ways that maintain these beliefs. That is, to protect against feared consequences, they engage in **safety behaviours**. For example, people who fear they will die from a fast heart rate stop all physical activity the minute they feel their heart race. They come to believe that only their safety behaviours have kept them alive. Hence, safety behaviours allow a person to maintain overly negative cognitions.

## **Perceived lack of control**

People who report experiencing little sense of control over their surroundings are at risk for a broad range of anxiety disorders (Mineka & Zinbarg, 1998). Childhood experiences, such as traumatic events (Green et al., 2010), punitive and restrictive parenting (Chorpita, Brown, & Barlow, 1998) or abuse (Chaffin, Silovsky, & Vaughn, 2005), may promote a view that life is not controllable. Similarly, anxiety disorders often develop after serious life events that threaten the sense of control over one’s life. Indeed, more than 70 percent of people report a severe life event before the onset of an anxiety disorder (Finlay-Jones, 1989). Other life experiences may shape the sense of control over the feared stimulus. For example, people who are used to dogs and feel comfortable about controlling dogs’ behaviour are much less likely to develop a phobia after a dog bite. On the whole, early and recent experiences of lack of control can influence whether a person develops an anxiety disorder (Mineka & Zinbarg, 2006).

Animal studies have illustrated that a lack of control over the environment can promote anxiety. For example, Insel and colleagues (1988) randomly assigned monkeys to one of two conditions. One group of monkeys grew up with the ability to choose whether and when they would receive treats. A second group of monkeys had no control over whether and when they received treats but received the same number of treats. In the third year of life, monkeys who had grown up without control behaved in ways that looked anxious when facing new situations and interacting with other monkeys; monkeys who had grown up with control showed less anxiety. In sum, animal and human studies both point towards the importance of perceived lack of control in the development of anxiety disorders.

## **Attention to threat**

A large body of research indicates that people with anxiety disorders pay more attention to negative cues in their environment than do people without anxiety disorders (Williams, Watts, MacLeod, & Mathews, 1997). In a meta-analysis of 172 studies, each of the specific anxiety disorders was associated with heightened attention to threatening stimuli on tasks such as the dot probe task, shown in figure 4.5 (Bar-Haim, Lamy, Pergamin et al., 2007). For example, people with social anxiety disorder have been found to selectively attend to angry faces (Staugaard, 2010), whereas people with snake phobias selectively attend to cues related to snakes (McNally, Caspi, Riemann, & Zeitlin, 1990; Öhman, Flykt, & Esteves, 2001). Researchers have also shown that this happens automatically and very quickly — before people are even consciously aware of the stimuli (Öhman & Soares, 1994; Staugaard, 2010). Once a threatening object captures their attention, anxious people have a difficult time pulling their attention away from that object; they tend to stay focused on a threatening object longer than others do (Cisler & Koster, 2010).

In one line of experimental research, investigators have examined whether attention to anxiety-related information can actually be created and then whether this attention ‘bias’ leads to more anxiety (MacLeod & Mathews, 2012). To train people to attend to threatening words, researchers used the dot probe task. To teach a negative bias, participants view hundreds of trials in which the dot is more likely to occur where the negative word was. For a control group, the dot is equally likely to appear where

either the negative or neutral word was. People who were trained to attend to negative words reported a more anxious mood after training, especially when they were given a challenging task like an unsolvable puzzle to perform. The control group did not show an increase in anxious mood after training. The findings suggest that the way we focus our attention can foster an anxious mood.

**FIGURE 4.5**

The computerised dot probe task is used to test biases in attention and, in some studies, to train attentional biases. On the first screen of each trial, participants see one neutral word and one negative word. On the second screen, a dot appears in the location where one of the two words was. The participant is asked to press a button as quickly as possible to indicate whether the dot appears on the left or right side of the screen. In the case shown here, a person who was looking at the word *death* will see the dot and respond more quickly than a person who was looking at the word *table*.

**First screen**



**Second screen**



Could these types of trained biases help us understand diagnosable levels of anxiety? Researchers have examined this question by training people diagnosed with generalised anxiety disorder to attend to positive information (Amir, Beard, Burns, & Bomyea, 2009). To train a positive bias, the researchers used a version of the dot probe task in which dots appeared where the positive words had been. Anxiety levels did not change over the course of the study in the control group. Participants in the positive-bias training condition obtained lower anxiety scores on self-report and interview measures post-training: 50 percent of the people who received positive-bias training no longer met the diagnostic criteria for GAD. Parallel benefits of attention training have been shown among people with social anxiety (Schmidt, Richey, Buckner, & Timpano, 2009). This type of training also has been shown to help reduce cortisol responses when people encounter daily stressors (Dandeneau, Baldwin, Baccus, Sakellaropoulou, & Pruessner, 2007).

## Aetiology of specific phobias

So far, we have discussed factors that might set the stage for development of anxiety disorders in general. Now, we turn to the question of how each of the specific anxiety disorders arises, beginning with phobias. That is, why does one person develop a specific phobia while another person develops generalised anxiety disorder?

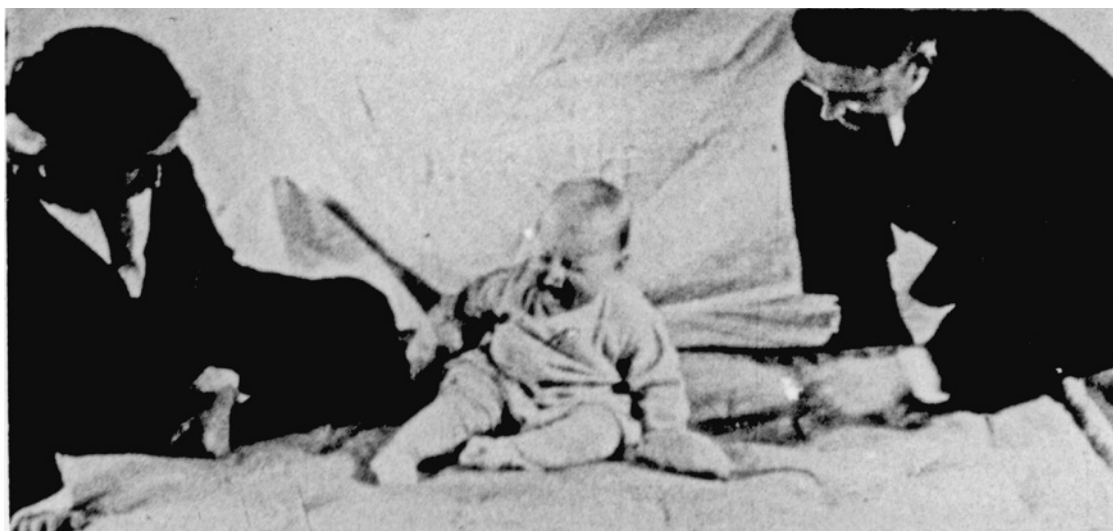
The dominant model of phobias is the two-factor model of behavioural conditioning, described earlier. In the behavioural model, specific phobias are seen as a conditioned response that develops after a threatening experience and is sustained by avoidant behaviour. In one of the first illustrations of this

model, John Watson and his graduate student Rosalie Rayner published a case report in 1920 in which they demonstrated creating an intense fear of a rat (a phobia) in an infant, Little Albert, using classical conditioning. Little Albert was initially unafraid of the rat, but after repeatedly seeing the rat while a very loud noise was made, he began to cry when he saw the rat.

As already mentioned, behavioural theory suggests that phobias *could* be conditioned by direct trauma, modelling or verbal instruction. In one study, 1937 people were asked whether they had these types of conditioning experiences before the onset of their phobias (Kendler, Myers, & Prescott, 2002). About half of the people in the study could not remember any such experiences. Obviously, if many phobias start without a conditioning experience, this is a big problem for the behavioural model. But proponents of the behavioural model argue that people may forget conditioning experiences (Mineka & Öhman, 2002).

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Little Albert, shown here with Rayner and Watson, was classically conditioned to develop a fear of a white rat.



Even among those who have had a threatening experience, many do not develop a phobia. How might we understand this? To begin with, the risk factors we have described, such as genetic vulnerability, neuroticism, negative cognition and propensity towards fear conditioning, probably operate as diatheses — vulnerability factors that shape whether or not a phobia will develop in the context of a conditioning experience (Mineka & Sutton, 2006).

It is also believed that only certain kinds of stimuli and experiences will contribute to development of a phobia. Mowrer's original two-factor model suggests that people could be conditioned to be afraid of all types of stimuli. But people with phobias tend to fear certain types of stimuli. Typically, people do not develop phobias of flowers, lambs or lamp shades! But phobias of insects or other animals, natural environments and blood are common. As many as half of women report a fear of snakes; moreover, many different types of animals also fear snakes (Öhman & Mineka, 2003). Researchers have suggested that during the evolution of our species, people learned to react strongly to stimuli that could be life-threatening, such as heights, snakes and angry humans (Seligman, 1971). Evolution may have 'prepared' our fear circuit to learn fear of certain stimuli very quickly and automatically; hence, this type of learning is called **prepared learning**. In support of this idea of evolutionarily adaptive fears, researchers have shown that monkeys can be conditioned to fear snakes and crocodiles but not flowers and rabbits (Cook & Mineka, 1989). As researchers have tested this model, some have discovered that people can be initially conditioned to fear many different types of stimuli (McNally, 1987). Fears of most types of stimuli fade quickly with ongoing exposure, though, whereas fears of naturally dangerous stimuli are sustained in most studies (Dawson, Schell, & Banis, 1986).

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The prepared learning model suggests that we have evolved to pay special attention to signs of danger, including angry people, threatening animals and dangerous natural environments.



## Aetiology of social anxiety disorder

In this section, we review behavioural and cognitive factors related to social anxiety disorder. The trait of behavioural inhibition may also be important in the development of social anxiety disorder.

### **Behavioural factors: conditioning of social anxiety disorder**

Behavioural perspectives on the causes of social anxiety disorder are similar to those on specific phobias, insofar as they are based on a two-factor conditioning model. That is, a person could have a negative social experience (directly, through modelling or through verbal instruction) and become classically conditioned to fear similar situations, which the person then avoids. The next step involves operant conditioning: avoidance behaviour is reinforced because it reduces the fear the person experiences. There are few opportunities for the conditioned fear to be extinguished because the person tends to avoid social situations. Even when the person interacts with others, he or she may show avoidant behaviour in smaller ways that have been labelled as safety behaviours. Examples of safety behaviours in social anxiety disorder include avoiding eye contact, disengaging from conversation and standing apart from others. Although these behaviours are used to avoid negative feedback, they create other problems. Other people tend to disapprove of these types of avoidant behaviours, which then intensifies the problem (Wells, 1998). (Think about how you might respond if you were trying to talk to someone who looks at the floor, fails to answer your questions and leaves the room in the middle of the conversation.)

### **Cognitive factors: too much focus on negative self-evaluations**

Theory focuses on several different ways in which cognitive processes might intensify social anxiety (Clark & Wells, 1995). First, people with social anxiety disorders appear to have unrealistically negative beliefs about the consequences of their social behaviours — for example, they may believe that others will reject them if they blush or pause while speaking. Second, they attend more to how they are doing in social situations and their own internal sensations than other people do. Instead of attending to their conversation partner, they are often thinking about how others might perceive them (e.g., ‘He must think I’m an idiot’). In one study, researchers asked men to recount the thoughts that ran through their mind when they thought about introducing themselves to an attractive woman (Zanov & Davison, 2010). Men with social anxiety were much more likely to verbalise thoughts about their own performance than were those without social anxiety (Bates, Campbell, & Burgess, 1990; Zanov & Davison, 2009). As they focus on their performance, they often form powerfully negative visual images of how others will react to them (Hirsch & Clark, 2004).

The evidence is clear that people with social anxiety disorder are overly negative in evaluating their social performance, even when they are not socially awkward (Stopa & Clark, 2000). For example, in one study, researchers assessed blushing in people with and without social anxiety disorder. Participants were asked to estimate how much they would blush during different tasks, such as singing a children's song. Then they were asked to engage in these different tasks. Participants with social anxiety disorder overestimated how much they would blush (Gerlach, Wilhelm, Gruber, & Roth, 2001). Similarly, one research team asked people with social anxiety disorder to rate videos of their performance in giving a short speech. Socially anxious people rated their speeches more negatively than objective raters did, whereas people who were not socially anxious were not harsh in rating their own performance (Ashbaugh, Antony, McCabe, Schmidt, & Swinson, 2005). The evidence is clear that people with social anxiety disorder are unfairly harsh in their self-evaluations.

There is also evidence that people with social anxiety disorder attend more to internal cues than to external (social) cues. For example, people with social anxiety disorder appear to spend more time than other people do monitoring for signs of their own anxiety. In one study, researchers gave participants a chance to watch their own heart rate displayed on a computer screen or to view faces. Many of the faces were threatening. People diagnosed with social anxiety disorder attended more closely to their own heart rate than did the people who were not diagnosed with social anxiety disorder (Pineles & Mineka, 2005). Hence, rather than keeping their eye on potential external threats, people with this disorder tend to be busy monitoring their own anxiety levels.

How might all these risk variables fit together when we consider a person with social anxiety disorder, like Maureen from the earlier clinical case? Maureen is likely to have inherited some tendency to be anxious when faced with new people. As she grew up, her anxiety may have interfered with her chances to acquire social skills and to gain self-confidence. Her fear of other people's opinions and her own negative thoughts about her social abilities created a vicious circle in which her intolerable anxiety led her to avoid social situations and then the avoidance led to increased anxiety. Even when she is in social situations, her focus on her performance and anxiety levels may interfere with being fully engaged in the interaction.

## Aetiology of panic disorder

In this section, we look at current thinking about the aetiology of panic disorder from neurobiological, behavioural and cognitive perspectives. As you will see, all of these perspectives focus on how people respond to somatic (bodily) changes like increased heart rate.

### Neurobiological factors

We have seen that the fear circuit appears to play an important role in many of the anxiety disorders. Now we will see that a particular part of the fear circuit is especially important in panic disorder: the **locus coeruleus** (see figure 4.6). The locus coeruleus is the major source of the neurotransmitter norepinephrine in the brain. Surges in norepinephrine are a natural response to stress and when these surges occur, they

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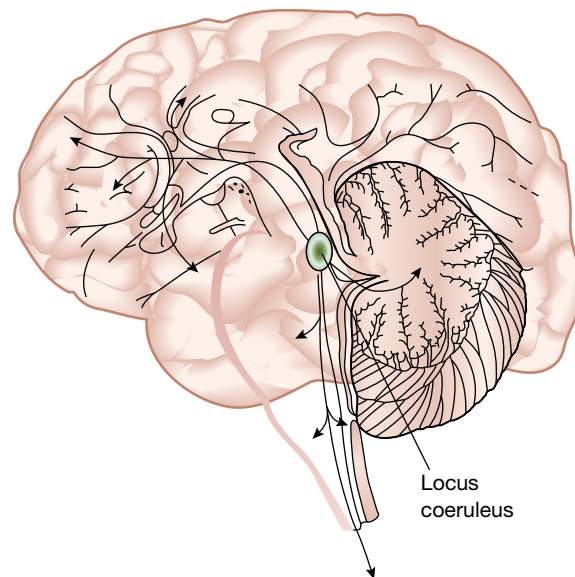
New Zealand actress Rebecca Gibney has spoken publicly about her battle with panic attacks ('Rebecca Gibney', 2014).



are associated with increased activity of the sympathetic nervous system, reflected in a faster heart rate and other psychophysiological responses that support the fight-or-flight response. People with panic disorder show a more dramatic biological response to drugs that trigger releases of norepinephrine (Neumeister, Daher, & Charney, 2005). Drugs that increase activity in the locus coeruleus can trigger panic attacks, and drugs that decrease activity in the locus coeruleus, including clonidine and some antidepressants, decrease the risk of panic attacks (Sullivan, Coplan, Kent, & Gorman, 1999).

**FIGURE 4.6**

The locus coeruleus is the major source of norepinephrine. Surges in norepinephrine lead to a number of physiological shifts, including faster heart rate.



*Source:* J. H. Martin, *Neuroanatomy Text and Atlas*, 4th ed. (1996), copyright McGraw Hill Education LLC with permission.

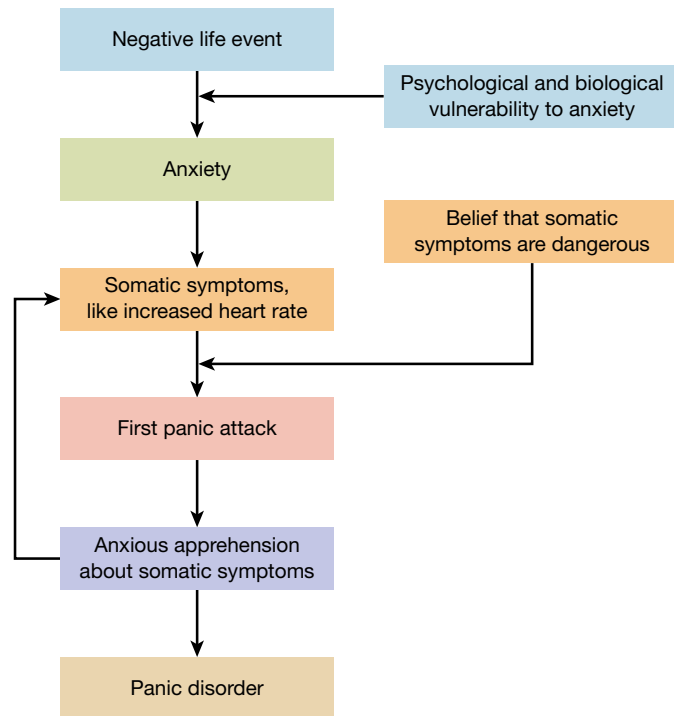
### **Behavioural factors: classical conditioning**

The behavioural perspective on the aetiology of panic disorder focuses on classical conditioning. This model draws from an interesting pattern — panic attacks are often triggered by internal bodily sensations of arousal (Kenardy & Taylor, 1999). Theory suggests that panic attacks are classically conditioned responses to either the situations that trigger anxiety or the internal bodily sensations of arousal (Bouton, Mineka, & Barlow, 2001). Classical conditioning of panic attacks in response to bodily sensations has been called **interoceptive conditioning**: a person experiences somatic signs of anxiety, which are followed by the person's first panic attack; panic attacks then become a conditioned response to the somatic changes (see figure 4.7).

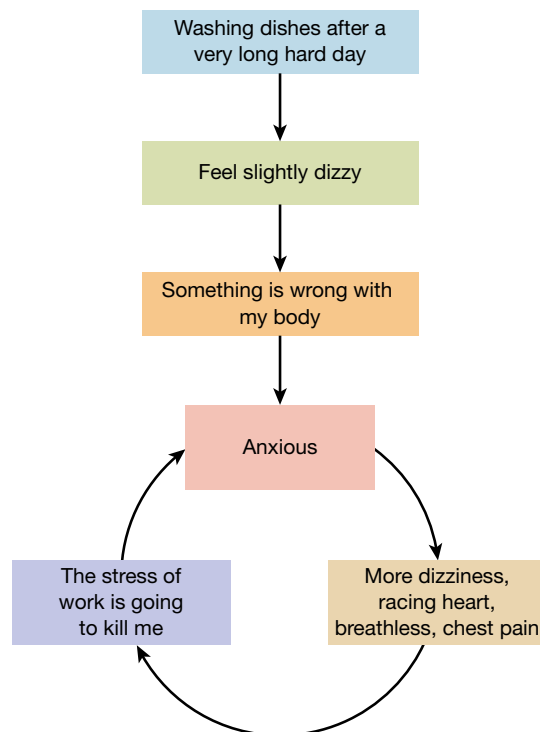
### **Cognitive factors in panic disorder**

Cognitive perspectives focus on catastrophic misinterpretations of somatic changes (Clark, 1996). According to this model, panic attacks develop when a person interprets bodily sensations as signs of impending doom (see figure 4.8). For example, the person may interpret the sensation of an increase in heart rate as a sign of an impending heart attack. Obviously, such thoughts will increase the person's anxiety, which produces more physical sensations, creating a vicious circle.

**FIGURE 4.7** Interoceptive conditioning



**FIGURE 4.8** An example of catastrophic misinterpretation of bodily cues



The evidence that these cognitive factors can contribute to panic attacks is quite strong. To understand the evidence, it is important to know that panic attacks can be experimentally induced in the laboratory. Research has focused on triggering panic attacks experimentally for more than 75 years. These studies suggest that an array of factors that create physiological sensations can trigger panic attacks among people with a history of panic attacks, including more than a dozen different medications (Swain, Koszycki, Shlik, & Bradwein, 2003). Even drugs that have opposite physiological effects can set off panic attacks (Lindemann & Finesinger, 1938). Exercise alone, simple relaxation or the physical sensations caused by an illness such as inner-ear disease also can induce panic attacks (Asmundson, Larsen, & Stein, 1998). Another commonly used procedure involves exposing people to air with high levels of carbon dioxide; in response to the diminished oxygen, breathing rate increases and for some people this induces panic. In short, many different bodily sensations can trigger panic attacks (Barlow, 2004). Cognitive researchers have focused on how to differentiate the people who do and do not develop a panic attack in these experimental studies. People who develop panic attacks after being exposed to these agents seem to differ from those who do not develop panic attacks on only one characteristic — the extent to which they are frightened by the bodily changes (Margraf, Ehlers, & Roth, 1986).

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Experiments demonstrate that panic attacks can be triggered by a variety of agents that change bodily sensations, including drugs and even exercise.



Consistent with the idea that people with panic disorder are more attuned to small signs of body changes, several studies suggest that when heart rate is monitored using psychophysiological equipment, people with panic disorder are more accurate than other people in knowing when an arrhythmia occurs and in detecting changes in their heart rate during periods of stress and arousal (Domschke, Stevens, Pfeleiderer, & Gerlach, 2010).

## Aetiology of agoraphobia

Because agoraphobia was only recognised as a distinct disorder with the DSM-5, less is known about its aetiology. As with other anxiety disorders, risk of agoraphobia appears to be related to genetic vulnerability and life events (Wittchen et al., 2010). One major model of how these symptoms evolves focuses on cognition.

The principal cognitive model for the aetiology of agoraphobia is the **fear-of-fear hypothesis** (Goldstein & Chambless, 1978), which suggests that agoraphobia is driven by negative thoughts about the consequences of experiencing anxiety in public. There is evidence that people with agoraphobia think the consequences of public anxiety would be horrible (Clark, 1997). They seem to have catastrophic beliefs that their anxiety will lead to socially unacceptable consequences, for example, 'I am going to go crazy'. (Chambless, Caputo, Bright, & Gallagher, 1984).

## Aetiology of generalised anxiety disorder

Generalised anxiety disorder (GAD) tends to co-occur with other anxiety disorders. Because the comorbidity is so high, researchers believe that many of the factors involved in predicting anxiety disorders in general are particularly important for understanding GAD. GAD, though, seems to differ from the other anxiety disorders in a few important ways. People who meet diagnostic criteria for GAD are much more likely to experience episodes of major depressive disorder than those with other anxiety disorders are. Moreover, GAD seems to involve a general tendency to experience general distress more than a specific pattern of intense fear. Indeed, whereas other anxiety disorders tend to be related to very intense psychophysiological responses to threatening stimuli, people diagnosed with GAD do not show this heightened psychophysiological response to threat (McTeague & Lang, 2012). Rather than fear, GAD is related to a more amorphous profile of general distress. Cognitive theory provides one way to make sense of the distress that is observed in GAD.

More specifically, cognitive factors may help explain why some people worry more than others. Worry is such an unpleasant emotion that one might ask why anyone would worry a lot (Borkovec & Newman, 1998). Borkovec and colleagues have marshalled evidence that worry is actually reinforcing because it distracts people from more powerful negative emotions and images. Worry does not involve powerful visual images and does not produce the physiological changes that usually accompany emotion. Rather, worry is more like a kind of repetitive self-talk about bad things that might happen but seems not to involve strong emotion. Indeed, worrying actually decreases psychophysiological signs of arousal (Freeston, Dugas, & Ladoceur, 1996). Thus, by worrying, people with GAD may be avoiding emotions that would be more unpleasant and more powerful than worry. But as a consequence of this avoidance, their underlying anxiety about these images does not extinguish.

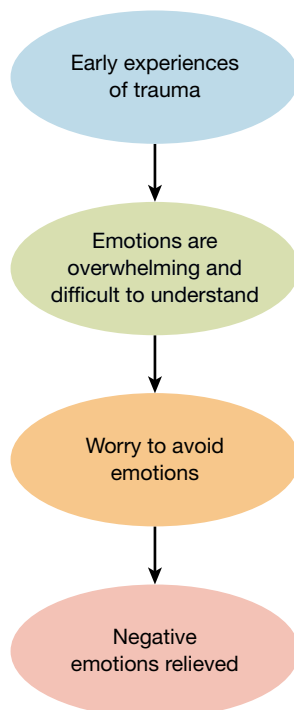
What kinds of anxiety-evoking images might people with GAD be avoiding? A possible answer comes from studies showing that many people with GAD report a history of traumatic experiences (Borkovec & Newman, 1998). In one study in which more than 1000 participants were followed from age 3 to 32, researchers coded various forms of childhood maltreatment, including maternal rejection, harsh discipline and childhood abuse. Maltreatment predicted a fourfold increase in the risk of developing GAD. It may be that worry distracts people with GAD from the distress of remembering these past traumas.

There is also support for the idea that people with GAD may be avoiding emotions. For example, people diagnosed with GAD report that they find it hard to understand and label their feelings (Mennin, Heimberg, Turk, & Fresco, 2002) and to regulate their negative emotions (Roemer et al., 2009) and they describe wanting to avoid intense emotion more than others do (Olatunji, Moretz, & Zlomke, 2010). See figure 4.9 for an overview of how these processes might contribute to worry.

Some research suggests that people who have a hard time accepting ambiguity are more likely to worry (that something bad *might* happen) and to develop GAD (Dugas, Marchand, & Ladouceur, 2005). This intolerance of uncertainty can predict increases in worry over time (Laugesen, Dugas, & Bukowski, 2003). Although intolerance of uncertainty is particularly strong for those with GAD, people with major depressive disorder and obsessive-compulsive disorder also tend to struggle when the future is uncertain (Gentes & Ruscio, 2011).

**FIGURE 4.9**

The excessive worry of GAD may be an attempt to avoid intense emotions.



## 4.4 Treatments of the anxiety disorders

**LEARNING OUTCOME 4.4** Describe treatment approaches that are common across the anxiety disorders and how treatment approaches are modified for the specific anxiety disorders.

Most people who seek treatment for anxiety disorders will only visit a family doctor. Most commonly, people will be offered a medication: in 2011, more than 80 million prescriptions were written in the United States for a class of anti-anxiety medications called benzodiazepines (IMS Health, 2012). As we shall see below, many other treatment options can be helpful for the anxiety disorders.

### Commonalities across psychological treatments

Effective psychological treatments for anxiety disorders share a common focus: exposure. Therapists from varying perspectives all agree that we must face up to the source of our fear or, as an ancient Chinese proverb puts it, 'Go straight to the heart of danger, for there you will find safety'. Even psychoanalysts, who

believe that the unconscious sources of anxiety are buried in the past, eventually encourage confronting the source of fears (Zane, 1984).

In a typical cognitive-behavioural approach to exposure treatment, the therapist and the client make a list of triggers — situations and activities that might elicit anxiety or fear — and create an ‘exposure hierarchy’, a graded list of the difficulty of these triggers. Early sessions involve exposure to relatively less challenging triggers and gradually, as the client learns that exposure will extinguish their anxiety, the more challenging triggers are faced. There have been more than 100 randomised controlled studies of cognitive-behavioural therapy (CBT) for anxiety disorders (Norton & Price, 2007) and dozens of these have compared CBT to a control treatment that involves some form of psychotherapy (Hofmann & Smits, 2008). Those studies suggest that CBT works well, even in comparison to other types of treatment. Exposure treatment is effective for 70–90 percent of clients.

The effects of CBT appear to endure when follow-up assessments are conducted six months after treatment (Hollon, Stewart, & Strunk, 2006) but in the years after treatment, many people experience some return of their anxiety symptoms (Lipsitz, Mannuzza, Klein, Ross, & Fyer, 1999). A couple of key principles appear important in protecting against relapse (Craske & Mystkowski, 2006). First, exposure should include as many features of the feared object as possible. For example, exposure for a person with a spider phobia might include a focus on the hairy legs, the beady eyes and other features of spiders. Second, exposure should be conducted in as many different contexts as possible (Bouton & Waddell, 2007). As an example, it might be important to expose a person to a spider in an office and also outside in nature.

The behavioural view of exposure is that it works by extinguishing the fear response. A good deal of work has focused on how extinction works at a neurobiological level and how this information might be used to refine exposure treatment (Craske et al., 2008). This work suggests that extinction does not work like an eraser. Let’s take dog phobia as an example. Extinction will not erase the underlying fear of dogs altogether — the conditioned fear still resides deep inside the brain and it can resurface over time or in certain contexts. Rather, extinction involves learning new associations to stimuli related to dogs. These newly learned associations inhibit activation of the fear. Thus, extinction involves learning, not forgetting.

A cognitive view of exposure treatment has also been proposed. In this view, exposure relieves symptoms by allowing people to realise that, contrary to their beliefs, they can tolerate aversive situations without loss of control (Foa & Meadows, 1997). Cognitive approaches to treatment of anxiety disorders typically focus on challenging (1) a person’s beliefs about the likelihood of negative outcomes if he or she faces an anxiety-provoking object or situation and (2) the expectation that he or she will be unable to cope. Thus cognitive treatments typically involve exposure in order to help people learn that they can cope with these situations. Because both behavioural and cognitive treatments involve exposure and learning to cope differently with fears, it is not surprising that most studies suggest that adding a cognitive therapy component to exposure therapy for anxiety disorders does not bolster results (Deacon & Abramowitz, 2004). Some very specific cognitive techniques, however, seem to help when added to exposure treatment, such as using web-based programs or virtual reality.

Although much of the work in this area is focused on exposure treatments, regardless of their format, several treatments have been developed more recently to help people take a more reflective, less reactive view of their intense anxiety and other emotions. These treatments include components such as mindfulness meditation and skills to promote acceptance of emotions, often in combination with some of the other CBT techniques. Although acceptance and mindfulness meditation treatments appear to be more powerful in reducing anxiety symptoms than are placebos, these treatments may not be as helpful as standard CBT approaches (Vøllestad, Nielsen, & Nielsen, 2012). Findings regarding how these approaches compare to CBT have varied and so more research is needed (Hofmann, Sawyer, Witt, & Oh, 2010).

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Virtual reality technology is sometimes used to facilitate exposure to feared stimuli.



### Medications that reduce anxiety

Drugs that reduce anxiety are referred to as **anxiolytics** (the suffix *lytic* comes from a Greek word meaning ‘to loosen or dissolve’). Two types of medications are most commonly used for the treatment of anxiety disorders: **benzodiazepines** (e.g., Valium and Xanax) and antidepressants, including tricyclic antidepressants, selective serotonin reuptake inhibitors (SSRIs) and **serotonin–norepinephrine reuptake inhibitors (SNRIs)** (Hoffman & Mathew, 2008). Benzodiazepines are sometimes referred to as minor tranquilisers or sedatives. A large number of studies have now confirmed that benzodiazepines and antidepressants provide more benefit than do placebos for anxiety disorders (Kapczinski, Lima, Souza, Cunha, & Schmitt, 2002; Moylan et al., 2011; Stein, Ipser, & Balkom, 2004). Beyond these medications that seem to help the range of anxiety disorders, certain drugs seem to be effective for specific anxiety disorders. For example, buspirone (BuSpar) has received approval from the US Food and Drug Administration for generalised anxiety disorder (Hoffman & Mathew, 2008).

Generally, antidepressants are preferred over benzodiazepines because people may experience severe withdrawal symptoms when they try to stop using

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Australian singer Missy Higgins has spoken publicly about being plagued with severe depression and anxiety.



benzodiazepines (Schweizer, Rickels, Case, & Greenblatt, 1990) — that is, they can be addictive. Benzodiazepines can have significant cognitive and motor side effects, such as memory lapses and drowsiness, and the side effects have been found to have real-world significance: benzodiazepines are related to an increased risk of car accidents (Rapoport et al., 2009). Antidepressants tend to have fewer side effects than benzodiazepines. Nonetheless, as many as half of people using tricyclic antidepressants discontinue this medication because of side effects such as jitteriness, weight gain, elevated heart rate and high blood pressure (Taylor et al., 1990). Compared to tricyclic antidepressants, SSRIs and SNRIs tend to have fewer side effects. As a result, SSRIs and SNRIs are considered the first-choice medication treatments of most anxiety disorders. Some people, however, do experience side effects from SSRIs and SNRIs, including restlessness, insomnia, headache and diminished sexual functioning (Bandelow et al., 2008). Many people stop taking anxiolytic medications because of the side effects.

This discussion leads us to the key problem: most people relapse once they stop taking medications. Because of this fact and because exposure treatments and medications provide comparable levels of relief from anxiety symptoms (Cuijpers et al., 2013), psychological treatments are considered the preferred treatment of most anxiety disorders, with the possible exception of GAD (Mitte, 2005).

## 4.5 Obsessive-compulsive and related disorders

**LEARNING OUTCOME 4.5** Define the symptoms and epidemiology of the obsessive-compulsive and related disorders and the trauma-related disorders.

### CLINICAL CASE

#### Jacob

Jacob was a 28-year-old postgraduate student when he sought treatment. Shortly after starting his postgraduate degree two years before, Jacob had begun to experience intrusive images of running over animals with his car. Although he knew that he had not done so, the images were so vivid that he could not get them out of his mind. Several times a week, he would be so disturbed by the idea that he could have hit animals that he would feel compelled to turn around and re-drive his route, looking for any signs of wounded or dead animals. If he spotted any, he would pull off the road and inspect his car for any signs that he might have been the person who hit the animal. Sometimes, when he finished his second trip, he would become concerned that he might have missed something and would feel the need to drive the route again. This often made him late for work or appointments, but if he didn't get the chance to look, he would ruminate about the possibility of injured animals throughout the day. He had a number of other thoughts and behaviours he felt compelled to do, including elaborate bathing rituals, maths problems that he solved repeatedly in his head, chewing his food a certain number of times, turning his light on and off three times before he left his bedroom and tapping his right elbow three times every hour. He knew these behaviours didn't make sense, but he still felt a terrible sense of anxiety if he didn't engage in them. The images occupied his mind and the behavioural rituals absorbed his time, such that his progress in his postgraduate degree had slowed to a crawl. Recently, his supervisor had raised concerns that he was not making sufficient progress in his thesis research.

#### QUESTION

What would be your initial assessment of Jacob? Why?

We will focus on three disorders in this section: obsessive-compulsive disorder (OCD), body dysmorphic disorder and hoarding disorder (see table 4.4). OCD, the prototypical disorder of this cluster, is defined by repetitive thoughts and urges (obsessions), as well as an irresistible need to engage in repetitive behaviours or mental acts (compulsions). Body dysmorphic disorder and hoarding disorder share symptoms of repetitive thoughts and behaviours. People with body dysmorphic disorder spend hours a day thinking about their appearance and almost all engage in compulsive behaviours related

to their appearance. People with hoarding disorder spend a lot of time repetitively thinking about their current and potential future possessions. They also engage in intensive efforts to acquire new objects and these efforts can resemble the compulsions observed in OCD. For all three conditions, the repetitive thoughts and behaviours are distressing, feel uncontrollable and require a considerable amount of time.

Beyond similarities in the symptoms, these syndromes often co-occur. About a third of people with body dysmorphic disorders, and up to a quarter of people with hoarding disorder, meet diagnostic criteria for OCD during their lifetime. About one-third of people with OCD experience at least some symptoms of hoarding (Steketee & Frost, 2003).

As you will see, people with these disorders report feeling anxious and they often experience other anxiety disorders as well (Brakoulias et al., 2011). Many of the risk factors for other anxiety disorders contribute to these disorders and the treatment approaches overlap. Nonetheless, these disorders also have some distinct causes compared to other anxiety disorders. As you read this section, it is important to consider parallels with the diagnoses described earlier in this chapter in relation to anxiety disorders. As we will see, there are many parallels in the aetiology and treatment of these three conditions (Phillips, et al., 2010).

**TABLE 4.4** Diagnoses of obsessive-compulsive and related disorders

DSM-5 diagnoses	Key features
Obsessive-compulsive disorder	Repetitive, intrusive, uncontrollable thoughts or urges (obsessions)  Repetitive behaviours or mental acts that the person feels compelled to perform (compulsions)
Body dysmorphic disorder	Preoccupation with imagined flaw in one's appearance  Excessive repetitive behaviours or acts regarding appearance (e.g., checking appearance, seeking reassurance)
Hoarding disorder	Acquisition of an excessive number of objects  Inability to part with those objects

## Clinical descriptions of the obsessive-compulsive and related disorders

As mentioned, the obsessive-compulsive and related disorders all share a quality of repetitive thought as well as irresistible urges to engage repetitively in some behaviour or mental act. As we will see next, though, the focus of thought and behaviour takes a different form across the three conditions.

### Obsessive-compulsive disorder

The diagnosis of **obsessive-compulsive disorder (OCD)** is based on the presence of obsessions or compulsions — most people with OCD experience both. Although many think that the chief distinction here is of thoughts (obsessions) versus behaviours (compulsions), they are mistaken.

**Obsessions** are intrusive and recurring thoughts, images or impulses that are persistent and uncontrollable and that often appear irrational to the person experiencing them. At least 80 percent of people experience brief intrusive thoughts from time to time — a terrible song or image gets stuck in your head (Rachman & DeSilva, 1978). And most of us also have urges now and then to behave in ways that would be embarrassing or dangerous. But few of us have thoughts or urges that are persistent and intrusive enough to qualify as obsessions. Like Jacob's images of animals (see the clinical case), the obsessions of

most people with OCD have such force and frequency that they interfere with normal activities. Typically, the person spends hours a day immersed in these thoughts, images or urges.

Obsessions often involve fear of contamination from germs or disease. Although most of us would like to avoid germs and disease, the fears of the person with OCD can be triggered by stimuli that are only remotely related to disease. One man with OCD was frightened by all cues that reminded him of cancer. He described seeing a bald cashier and thinking that the man's baldness might have been the result of chemotherapy. He stopped shopping at the store lest he be exposed to whatever toxins could have contributed to the man's cancer (Woody & Teachman, 2000). Others with contamination fears describe the need to change clothes and shower after being in a room with someone who has coughed and then to wash the clothes as well as any part of their home that the contaminated clothes might have touched.

Other frequent foci for obsessions include sexual or aggressive impulses, body problems, religion, and symmetry or order (Bloch et al., 2008). Those with concerns about aggressive impulses might be haunted by disturbing images of hurting their loved ones, despite no anger or desire to do so. Terrified by a recurrent image of throwing her newborn baby down the stairs, one woman with OCD avoided standing on stairs for months (Belluck, 2014).

**Compulsions** are repetitive, clearly excessive behaviours or mental acts that the person feels driven to perform to reduce the anxiety caused by obsessive thoughts or to prevent some calamity from occurring. Jacob's need to revisit his route, described in the clinical case, fits this definition. Many people with OCD feel compelled to repeat a ritual if they did not execute it with precision. The sheer frequency with which compulsions are repeated may be staggering. Commonly reported compulsions include:

- pursuing cleanliness and orderliness, sometimes through elaborate rituals, such as showering for hours, wiping down all objects as they enter the house or asking visitors to wash before they enter the house
- performing repetitive, magically protective acts, such as counting, touching a body part, solving a maths problem or repeating a word again and again
- repetitive checking, such as returning seven or eight times in a row to see that lights, stove burners or taps are turned off, windows fastened and doors locked.

We often hear people described as compulsive gamblers, compulsive eaters and compulsive drinkers. Even though people may report irresistible urges to gamble, eat and drink, clinicians do not regard these behaviours as compulsions because they are often experienced as pleasurable. In contrast, most people view their compulsive rituals as 'rather silly or absurd', even though they are unable to stop performing them (Stern & Cobb, 1978). Although they may find their compulsions to be illogical, it is extremely difficult to stop — people with OCD feel as though something dire will happen if they do not perform the act.

OCD tends to begin either before age 10 or else in late adolescence/early adulthood (Conceicao do Rosario-Campos et al., 2001). It has been described in children as young as two years old (Rapoport, Swedo, & Leonard, 1992). Longitudinal research suggests that for most people, symptoms tend to

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For people with obsessive-compulsive disorder, extreme fears of contamination can trigger abnormally frequent handwashing.



be fairly chronic (Eisen et al., 2013). For those diagnosed with OCD, the pattern of symptoms appears to be similar across cultures (Seedat & Matsunaga, 2007). In addition to obsessions and compulsions, people with OCD are prone to extreme doubts, procrastination and indecision.

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Australian boxer Luke Jackson has described his daily fight with OCD, and the ways in which it both hinders and helps his training. He encourages others with the disorder to seek help in managing it (Rääbus, 2016).



#### DSM-5

##### **DSM-5 criteria for obsessive-compulsive disorder**

- People with obsessive-compulsive disorder experience obsessions and/or compulsions.
- Obsessions are defined by:
  - recurrent, intrusive, persistent, unwanted thoughts, urges or images
  - the person tries to ignore, suppress or neutralise the thoughts, urges or images.
- Compulsions are defined by:
  - repetitive behaviours or thoughts that the person feels compelled to perform to prevent distress or a dreaded event
  - the person feels driven to perform the repetitive behaviours or thoughts in response to obsessions or according to rigid rules.
- The acts are excessive or unlikely to prevent the dreaded situation.
- The obsessions or compulsions are time consuming (e.g., at least one hour per day) or cause clinically significant distress or impairment.

## RESEARCH EXAMPLE

The concept of a sense of control has long been suspected as central to anxiety and mood disorders. This is thought by some to be particularly relevant to obsessive-compulsive disorder. However, contemporary theories of OCD focus only on a need to control thoughts, rather than on aspects of controlling the self (behaviour as well as thoughts) and the external world.

A review by Moulding and Kyrios (2006) of research into control-related beliefs about OCD suggests that the relationship between a person's *desired* level of control and their *perceived* level of control could be a contributing factor in the development of OCD symptoms, including the superstitious aspect of their ritualised behaviours. The study suggested that further consideration of the place of desired and perceived levels of control could increase understanding and treatment of OCD.

### QUESTION

Do you agree with Moulding and Kyrios' findings? Why/why not?

## Body dysmorphic disorder

People with **body dysmorphic disorder (BDD)** are preoccupied with one or more imagined or exaggerated defects in their appearance. Although people with BDD may appear attractive to others, they perceive themselves as ugly or even 'monstrous' in their appearance (Phillips, 2006). Women tend to focus on their skin, hair, facial features, hips, breasts and legs, whereas men are more likely to focus on their height, penis size or body hair (Perugi, Akiskal, Giannotti et al., 1997). Some men suffer from the preoccupation that their body is small or insufficiently muscular, even when others would not share this perception.

The concerns of people with BDD have an obsessional flavour in that they find it very hard to stop thinking about their concerns. As one woman wrote: 'It's always in the back of my mind. I can't push it away. It's always there, taunting and haunting me' (Phillips, 2005, p. 69). On average, people with BDD think about their appearance for 3 to 8 hours per day (Phillips, Wilhelm et al., 2010). Also, like people with OCD, people with BDD find themselves compelled to engage in certain behaviours, such as checking their appearance in the mirror, comparing their appearance to that of other people, asking others for reassurance about their appearance, or using strategies to change their appearance or camouflage disliked body areas (grooming, tanning, exercising, changing clothes and applying makeup) (Phillips, Wilhelm et al., 2010). While many spend hours a day checking their appearance, some try to avoid being reminded of their perceived flaws by avoiding mirrors, reflective surfaces or bright lights (Phillips, 2005). Although most of us do things to feel better about our appearance, people with this disorder spend an inordinate amount of time and energy on these endeavours.

The symptoms are extremely distressing. About a third of patients with BDD have little insight into their overly harsh views and so they are convinced that others will see their flaws as grotesque (Phillips et al., 2012). As many as a fifth endure plastic surgery and many withstand multiple surgeries (Phillips, 2005). Unfortunately, plastic surgery does so little to allay their concerns that many report wanting to sue or hurt their physicians after the surgery (Veale, 2000). About a third of people with BDD endorse some history of suicidal ideation and about 20 percent have attempted suicide (Buhlmann et al., 2010).

## CLINICAL CASE

### Paul

After years of living with anxiety and shame over his appearance, Paul sought psychotherapy at age 33. He had first become 'horrified' by his appearance during puberty when he noticed that he was not developing the square jaw lines that many of his male friends had begun to show. In the past several

years, his shame was more focused on his nose, which he perceived as being too thin. He had sought surgery, but the surgeon refused to operate on his seemingly flawless nose. Over the past two decades, his preoccupation with his appearance had frequently interfered with socialising and altogether obstructed his ability to date. Even when he did venture out, he had to check his appearance repeatedly throughout the evening. Sometimes he was unable to go to work in his position as a physical therapist because he was overwhelmed by his anxiety. When he did go to work, he worried that his clients were too distracted by his physical flaws to attend to his instructions. (Wilhelm, Buhlmann, Hayward, Greenberg, & Dimaite, 2010)

### QUESTIONS

1. In your opinion, at what point does concern over physical appearance become pathological?
2. What DSM-5 criteria for BDD does Paul display?

As is the case for Paul (see the clinical case), preoccupation with appearance can interfere with many aspects of occupational and social functioning. To cope with the intense shame they feel about their appearance, people with BDD may avoid contact with others. In one survey, about a third reported missing work or avoiding school in the past month due to concerns about their appearance (Phillips, 2005). In another survey, about 40 percent of people with the disorder reported being unable to work (Didie, Menard, Stern, & Phillips, 2008) and some even become housebound.

BDD typically begins in adolescence. About 90 percent of persons diagnosed with the disorder still report symptoms four years after diagnosis (Phillips, Menard, Quinn, Didie, & Stout, 2013). Many people never receive treatment, in part because mental health professionals often do not ask about these symptoms and in part because those with the disorder often feel too ashamed to raise their concerns (Phillips, 2005).

Social and cultural factors surely play a role in how people decide whether they are attractive. Among university students, concerns about body appearance appear to be more common in America than in Europe — as many as 74 percent of American students report at least some concern about their body image, with women being more likely than men to report dissatisfaction (Bohne, Keuthen, Wilhelm, Deckersback, & Jenike, 2002). Most of these concerns, though, are not extreme enough to be characterised as psychological disorders. People with BDD experience agonising distress over their perceived physical flaws.

Among those who develop BDD, case reports from around the world suggest that the symptoms and outcomes are similar across cultures (Phillips, 2005). The body part that becomes a focus of concern sometimes differs by culture, though. For example, eyelid concerns are more common in Japan than in Western countries. In addition, Japanese patients with BDD appear to be more concerned about offending others than are Western patients (Suzuki, Takei, Kawai, Minabe, & Mori, 2003).

### DSM-5

#### DSM-5 criteria for body dysmorphic disorder

- People with body dysmorphic disorder experience preoccupation with one or more perceived defects in appearance.
- Others find the perceived defect(s) slight or unobservable.
- The person has performed repetitive behaviours or mental acts (e.g., mirror checking, seeking reassurance or excessive grooming) in response to the appearance concerns.
- Preoccupation is not restricted to concerns about weight or body fat.

Care should be taken to distinguish BDD from eating disorders. Most people with BDD are concerned about several different aspects of their appearance. When shape and weight concerns are the only foci, however, clinicians should consider whether the symptoms are better explained by an eating disorder.

## Hoarding disorder

Collecting is a favourite hobby for many people and almost all children go through a phase of collecting objects, but these hobbies are distinctly different from hoarding. Undoubtedly, the desire to acquire and save objects varies along a continuum (Timpano et al., 2013). For people with **hoarding disorder**, the need to acquire is clearly excessive and they abhor parting with their objects, even when others cannot see any potential value in them. Most typically, as illustrated in the clinical case below of Dena, the person has acquired a huge range of different kinds of objects — collections of clothes, tools or antiques may be gathered along with old containers and bottle caps.

### DSM-5

#### DSM-5 criteria for hoarding disorder

- People who meet the criteria for hoarding disorder experience persistent difficulty discarding or parting with possessions, regardless of their actual value.
- People with hoarding disorder experience a perceived need to save items and distress associated with discarding.
- The symptoms result in the accumulation of a large number of possessions that clutter active living spaces to the extent that their intended use is compromised unless others intervene.

Many people who hoard are unaware of the severity of their behaviour (Steketee & Frost, 2003), but to those surrounding them, the consequences of hoarding are clear and sometimes seem quite severe. The accrual of objects often overwhelms the person's home. In one study, case workers for elder services agencies were asked to describe their clients who suffered from hoarding disorder. Although the sampling strategy likely focused on particularly severe cases, the descriptions were notable. The case workers reported that the hoarding led to extremely filthy homes for about a third of their clients who had problems with hoarding, characterised by overpowering odours from rotten food or faeces. More than 40 percent had accumulated so many items that they were no longer able to use their refrigerator, kitchen sink or bathtub, and about 10 percent were unable to use their toilet (Kim, Steketee, & Frost, 2001). The poor hygiene, exposure to dirt and difficulties with cooking can all contribute to poor physical health, such as respiratory problems. Many family members sever relationships, unable to understand the attachment to the objects. About three-quarters of people with hoarding disorder engage in excessive buying (Frost, Tolin, Steketee, Fitch, & Selbo-Bruns, 2009) and many are unable to work (Tolin, Frost, Steketee, Gray, & Fitch, 2008), making poverty all too common among people with this condition (Samuels et al., 2007). As the problem escalates, health and safety officials often become involved. About 10 percent of persons with hoarding disorder will be threatened with eviction at some point in their lives (Tolin et al., 2008). For some, the money spent on acquiring possessions leads to homelessness.

About one-third of people with hoarding disorder, much more often women than men, also engage in animal hoarding (Patronek & Nathanson, 2009). People who engage in animal hoarding sometimes view themselves as animal rescuers, but those who witness the problem see it differently — the accumulating number of animals often outstrips the person's ability to provide adequate care, shelter and food. Animal hoarding is more likely to be associated with squalor than are other forms of hoarding. Animal protection agencies sometimes become involved.

Hoarding behaviour usually begins in childhood or early adolescence (Grisham, Frost, Steketee, Kim, & Hood, 2006). These early symptoms may be kept under control by parents and by limited income, so severe impairment from the hoarding often does not surface until later in life. Animal hoarding often does not emerge until middle age or older (Patronek & Nathanson, 2009).

Hoarding captured the attention of the public in 1947, when the Collyer brothers were found dead, surrounded by 140 tons of objects, ranging from grand pianos to antique sculptures to a human skeleton, piled from floor to ceiling in their New York City brownstone.



#### CLINICAL CASE

##### Dena

Dena was referred for treatment after animal care officials received reports from neighbours. A home inspection revealed over 100 animals living in her 1.2 hectare backyard and inside her house, many of them suffering from malnutrition, overcrowding and disease. When interviewed, she reported that she was running a rescue mission for animals and that she was 'just a little behind' because donations had diminished.

When the therapist visited her home, it became clear that her collections extended far beyond her animals. The rooms of her small home were so crowded that two of the doors to the outside could no longer be reached. Heaps of clothes and fabric mixed with miscellaneous furniture parts brimmed to the ceiling of her living room. In the kitchen, a collection of theatre memorabilia crowded out access to the stove and refrigerator. The dining room was covered with assorted items — bags of rubbish, piles of bills, stacks of old newspapers and several sets of china she had purchased 'at a bargain' at garage sales.

When the therapist offered to help Dena organise and neaten her home, Dena became enraged. She said that she had only allowed the therapist to visit to help her come to an arrangement with the animal control authorities and that she did not want to hear any comments about her home. She described years of fighting with her family over her housekeeping and she said she had done everything in her power to escape from their uptight rules and expectations. She denied needing the stove, stating that she wasn't about to start cooking hot meals as a single woman living alone. After the initial unsuccessful home visit, she refused any further contact with the therapist.

### QUESTIONS

1. What could be some of the next steps to get Dena the help she needs?
2. What are some of the health concerns you would have for Dena?

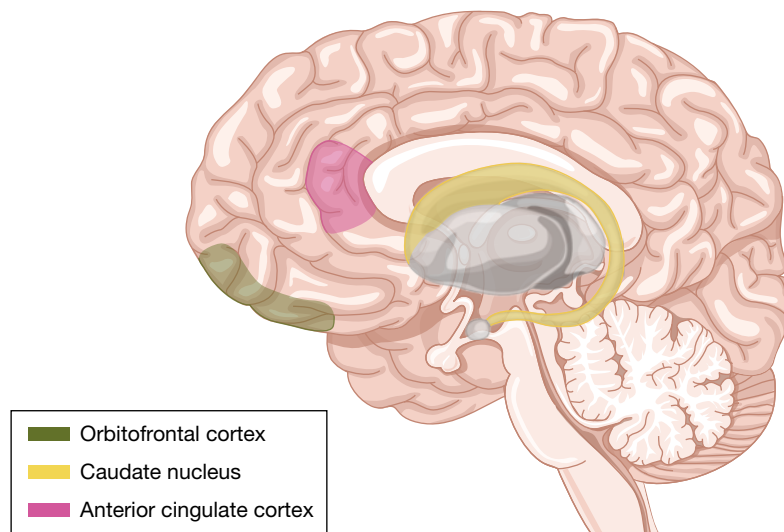
## 4.6 Aetiology of the obsessive-compulsive and related disorders

**LEARNING OUTCOME 4.6** Describe the commonalities in the aetiology of obsessive-compulsive and related disorders, as well as the factors that shape the expression of the specific disorders within this cluster.

There is a moderate genetic contribution to OCD, hoarding and body dysmorphic symptoms. Heritability is estimated to account for 40 to 50 percent of the variance in whether each of these conditions develops (Iervolino et al., 2009; Monzani, Rijdsdijk, Iervolino et al., 2012; Taylor, Jang, & Asmundson, 2010).

Obsessive-compulsive disorder, body dysmorphic disorder and hoarding disorder share some overlap in aetiology that is particularly apparent for genetic and neurobiological risk factors. For example, people with BDD and hoarding disorder often have a family history of OCD (Gustad & Phillips, 2003; Taylor et al., 2010), which could be the result of a shared genetic vulnerability. In regard to neurobiological risk, OCD, BDD and hoarding disorder seem to involve some of the same brain regions. Brain-imaging studies indicate that three closely related areas of the brain are unusually active in people with OCD (see figure 4.10): the **orbitofrontal cortex** (an area of the medial prefrontal cortex located just above the eyes), the **caudate nucleus** (part of the basal ganglia) and the anterior cingulate (Menzies et al., 2008; Rotge et al., 2009). When people with OCD are shown objects that tend to provoke symptoms (such as a soiled glove for a person who fears contamination), activity in these three areas increases (McGuire, Bench, Frith, & Marks, 1994). A similar pattern emerges when people with BDD view pictures of their own face. In those studies, BDD appears to be related to hyperactivity of the orbitofrontal cortex and the caudate nucleus (Feusner, Phillips, & Stein, 2010). When people with hoarding disorder are faced with decisions about whether to keep or discard possessions such as old mail, they show hyperactivity in the orbitofrontal cortex (Tolin, Kiehl, Worhunsky, Book, & Maltby, 2009) and the anterior cingulate compared to a control group (Tolin et al., 2012).

**FIGURE 4.10** Key brain regions in the obsessive-compulsive and related disorders: the orbitofrontal cortex, caudate nucleus and anterior cingulate cortex



While these genetic and neurobiological risk factors may set the stage for developing one of these disorders, why might one person develop OCD and another develop body dysmorphic disorder? Cognitive-behavioural models focus on factors that might promote one disorder as compared to the other.

## Aetiology of obsessive-compulsive disorder

Cognitive-behavioural models have been developed to explain how a person might get stuck repeating obsessions and compulsions again and again. A narrower cognitive model focuses on why obsessions might persist.

### Cognitive-behavioural models of obsessions and compulsions

In moderation, many of the thoughts and behaviours that disrupt the days of those with OCD, such as checking, cleaning or reconsidering a thought, have adaptive value. Cleaning, for example, can help reduce the risk of contamination and germs. The central goal of cognitive-behavioural theory, then, is to understand why the person with OCD continues to show the behaviours or thoughts used to ward off an initial threat well after that threat is gone.

To gather data on how people with OCD respond once a threat is gone, researchers conducted a two-phase experiment. In the first phase, they created a threat by placing electrodes on participants' wrists and then teaching participants that they would receive a shock (an unconditioned stimulus) when a certain shape (the conditioned stimulus) appeared on the computer screen. To avoid the shock, participants had to press a foot pedal (the conditioned response). In this first phase, participants with and without OCD learned equally well to press the foot pedal to avoid shock. In the key second phase of the study, researchers unhooked the wrist electrodes so that the participants could see that the threat of shock was gone. Even though they knew the threat was removed and even showed little psychophysiological response to the stimulus, many people with OCD either pressed the foot pedal or felt a strong urge to press it when the conditioned stimulus (the shape) appeared on the screen. People without OCD stopped pressing the foot pedal and most didn't have an urge to press it. The authors argue that for those with OCD, previously functional responses for reducing threat had become habitual and hence difficult to override after the threat was gone (Gillan et al., 2014). Consistent with this idea, other researchers have found that once someone with OCD develops a conditioned response to a stimulus, they are slower to change their response to that stimulus after it is no longer rewarded (Voon et al., 2014).

One cognitive model fits well with the idea that people with OCD continue to engage in behaviours to ward off threat, even after they can state that the threat is gone. According to this model, OCD is related to a deficit in the intuitive sense of feeling security and closure. Consider for a moment how we know to stop thinking about something or to quit studying for a test or organising our desk. There is no absolute signal from the environment. Rather, most of us stop when we have the sense of 'that is enough'. **Yedasentience** is defined as this subjective feeling of knowing that you have thought enough, cleaned enough or in other ways done what you should to prevent chaos and danger from low-level threats in the environment (Woody & Szechtman, 2011). It is theorised that people with OCD suffer from a biologically based deficit in yedasentience. Because they fail to gain the intuitive sense of completion and security, they have a hard time stopping their thoughts and behaviours. Objectively, they know that there is no need to check the stove or wash their hands again, but they suffer from an anxious internal sense that things are not complete. Compulsions are particularly reinforcing because they help relieve this sensation and they do so even more effectively than self-statements do (Woody & Szechtman, 2011).

### Thought suppression: a cognitive model of obsessions

A different model focuses on obsessions. This model suggests that people with OCD may try harder to suppress their obsessions than other people and, in doing so, may actually make the situation worse. Several researchers have shown that people with OCD tend to believe that thinking about something can make it more likely to occur (Rachman, 1977). People with OCD are also likely to describe especially deep feelings of responsibility for what occurs (Ladoceur et al., 2000). As a consequence of these two factors, they

are more likely to attempt **thought suppression** (Salkovskis, 1996). Consistent with this theory, people with OCD report engaging in thought suppression more than others do (Amir, Cashman, & Foa, 1997).

Unfortunately, it is hard to suppress thoughts. Consider the findings of one study of what happens when people are asked to suppress a thought (Wegner, Schneider, Carter, & White, 1987). Two groups of university students were asked either to think about a white bear or not to think about one. Attempts to avoid thinking about the white bear did not work — students thought about the bear more than once a minute when trying not to do so. Beyond that, there was a rebound effect — after students tried to suppress thoughts about the bear for five minutes, they thought about it much more often during the next five minutes. Trying to suppress a thought may have the paradoxical effect of inducing preoccupation with it. Indeed, in one experimental study, suppressing thoughts even briefly led to more intrusions of that thought over the next four days (Trinder & Salkovskis, 1994).

## Aetiology of body dysmorphic disorder

Cognitive models of BDD focus on what happens when a person with this syndrome looks at his or her body. People with BDD can accurately see and process their physical features — the problem does not appear to be one of distortion of the physical features. Rather, people with BDD tend to be detail oriented and this influences how they look at facial features (Feusner et al., 2010). Instead of considering the whole, they examine one feature at a time, which makes it more likely that they will become engrossed in considering a small flaw (Lambrou, Veale, & Wilson, 2011). They also consider attractiveness to be vastly more important than do control participants (Lambrou et al., 2011). Indeed, many people with BDD seem to believe that their self-worth is exclusively dependent on their appearance (Veale, 2004).

## Aetiology of hoarding disorder

In considering hoarding, many take an evolutionary perspective (Zohar & Felz, 2001). Imagine you were a caveman with no access to supermarkets to replenish food reserves and no clothing stores to find warm clothes when the weather got cold. In those situations, it would be adaptive to store any resources you could find. The question, though, is how these basic instincts become so uncontrollable for some people. The cognitive-behavioural model suggests a number of factors that might be involved. According to this model, hoarding is related to poor organisational abilities, unusual beliefs about possessions and avoidance behaviours (Steketee & Frost, 2003).

People with hoarding disorder have several different types of problems with cognitive organisational abilities. Problems with attention interfere with staying focused on the task at hand (Tolin & Villavicencio, 2011) and even once they do focus on dealing with their possessions, they have difficulty categorising objects and making decisions (Samuels, 2009). When asked to sort objects into categories in laboratory studies, people with hoarding disorder tend to be slow, to generate many more categories than others do and to find the process highly anxiety-provoking (Wincze, Steketee, & Frost, 2007). In their daily lives, faced with decisions about which object is the better one to acquire, many will go ahead and get two, three or more of the same type of object. Many patients find it excruciatingly hard to sort through their objects and figure out what to discard, even with a supportive therapist present (Frost & Steketee, 2010).

Beyond these difficulties with organisational abilities, the cognitive model focuses on the unusual beliefs that people with hoarding disorder hold. Almost by definition, people with hoarding disorder demonstrate an extreme emotional attachment to their possessions. They report feeling comforted by their objects, being frightened by the idea of losing an object and seeing the objects as core to their sense of self and identity. They hold a deep sense of responsibility for taking care of those objects and are likely to resent it if others touch, borrow or remove them (Steketee & Frost, 2003). Many feel a sense of grief when forced to part with an object (Frost, Steketee, & Tolin, 2012). These attachments may be even stronger when animals are involved. People who hoard animals often describe their animals as their closest confidants (Patronek & Nathanson, 2009). These beliefs about the importance of each and every object interfere with any attempt to tackle the clutter.

In the face of the anxiety of all these decisions, avoidance is common. Many people with this disorder find organising their clutter so overwhelming that they delay tackling the chaos (Frost, Steketee, & Greene, 2003). Avoidance is considered one of the key factors that maintains the clutter.

## 4.7 Treatment of the obsessive-compulsive and related disorders

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**LEARNING OUTCOME 4.7** describe the medication and psychological treatments for the obsessive-compulsive and related disorders

Treatments that work for obsessive-compulsive disorder, body dysmorphic disorder and hoarding disorder are similar. Each of these disorders responds to antidepressant medications. The major psychological approach is exposure and response prevention, although this treatment is tailored for the specific conditions.

### Medications

Antidepressants are the most commonly used medications for the obsessive-compulsive and related disorders. Although they were initially developed to treat depression, randomised controlled trials support their effectiveness in the treatment of OCD (Steketee & Barlow, 2004) and BDD (Grant, Odlaug, & Schreiber, 2014).

Only two randomised controlled trials of medication treatment have been conducted for BDD. Both trials found that antidepressants were more helpful than placebo in reducing symptoms. These trials have provided support for both clomipramine and fluoxetine as treatments of BDD (Ipser, Sander, & Stein, 2009). As with OCD, many people continue to experience at least mild symptoms of BDD with antidepressant treatment.

No randomised controlled trials of medications are available for hoarding disorder. Much of our knowledge is based on studies of OCD patients who also have hoarding symptoms. Although most studies indicate that hoarding symptoms respond less to medication treatment than do other OCD symptoms (Steketee & Frost, 2003), findings of one study indicated that patients with hoarding disorder demonstrated as much of a response to an antidepressant as did those with OCD (Saxena, Brody, Maidment, & Baxter, 2007).

### Psychological treatment

The most widely used psychological treatment for the obsessive-compulsive and related disorders is **exposure and response prevention (ERP)**. Victor Meyer (1966) developed this approach for OCD by tailoring the exposure treatment to address the compulsive rituals that people with OCD use to ward off threats.

#### Obsessive-compulsive disorder

Many OCD sufferers hold an almost magical belief that their compulsive behaviour will prevent awful things from happening. In the response prevention component of ERP, people expose themselves to situations that elicit the compulsive act and then refrain from performing the compulsive ritual — for instance, the person touches a dirty dish and then refrains from washing his or her hands. The reasoning behind this approach goes like this.

1. Not performing the ritual exposes the person to the full force of the anxiety provoked by the stimulus.
2. The exposure promotes the extinction of the conditioned response (the anxiety).

The exposure component of ERP uses the exposure hierarchy approach that we discussed earlier, in which a client begins with tackling less threatening stimuli and progresses to completing exposure sessions focused on more threatening stimuli. As an example, a client with a focus on contamination might start by being in a dirty room and progress to placing their hands on a sticky, grimy floor. Throughout the exposure sessions, the client is guided to avoid engaging in compulsions. Often, the therapist guides

exposure to feared stimuli in the home, with help from family members (Foa & Franklin, 2001). Typically, ERP involves refraining from performing rituals during therapy sessions lasting up to 90 minutes as well as during home practice between sessions.

Refraining from performing a ritual is extremely unpleasant for people with OCD. (To get some idea of how unpleasant, try delaying for a minute or two before scratching an itch.) Given the intensity of treatment, it is not surprising that about 25 percent of clients refuse ERP treatment (Foa & Franklin, 2001).

A meta-analysis of 19 studies comparing ERP to control treatments suggested that ERP is highly effective in reducing obsessions and compulsions (Rosa-Alcazar, Sanchez-Meca, Gomez-Conesa, & Marin-Martinez, 2008). ERP is more effective than clomipramine for the treatment of OCD (Foa et al., 2005) and it is effective for children and adolescents as well as adults (Franklin & Foa, 2011). Researchers have shown excellent outcomes for ERP offered by community therapists who do not specialise in OCD (Franklin & Foa, 2011). More than three quarters of people who receive this treatment show significant improvement (Mancebo, Eisen, Sibrava, Dyck, & Rasmussen, 2011), although mild symptoms often persist.

Cognitive approaches to OCD focus on challenging people's beliefs about what will happen if they do not engage in rituals (Van Oppen et al., 1995) or challenging their often inflated sense of responsibility (Clark, 2006). Eventually, to help test such beliefs, these approaches will use exposure. Several studies suggest that cognitive approaches perform as well as ERP (DeRubeis & Crits-Christoph, 1998).

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Exposure treatment for OCD involves confronting one's worst fears, such as contamination by dirty objects.



### **Body dysmorphic disorder**

The basic principles of ERP are tailored in several ways to address the symptoms of BDD. For example, to provide exposure to the most feared activities, clients might be asked to interact with people who could be critical of their looks. For response prevention, clients are asked to avoid activities they engage in to reassure themselves about their appearance, such as looking in mirrors. These behavioural techniques are supplemented with strategies to address the cognitive features of the disorder, such as the excessively critical evaluations of physical features and the belief that self-worth depends on appearance.

Several trials have shown that ERP produces a major decrease in body dysmorphic symptoms compared to control conditions (Wilhelm et al., 2014). Treatments that include a cognitive component may be more powerful than those that address only behaviours (Williams, Hadjistavropoulos, & Sharpe, 2006), but both treatments produce lasting effects (Ipser et al., 2009).

### **Hoarding disorder**

Treatment for hoarding is based on an adaptation of the ERP therapy that is employed with OCD (Steketee & Frost, 2003). The exposure element of treatment focuses on the most feared situation for people with hoarding disorder — getting rid of their objects. Response prevention centres on halting the rituals that they engage in to reduce their anxiety, such as counting or sorting their possessions. As with other exposure treatments, the client and the therapist work through a hierarchy, tackling increasingly difficult challenges as therapy progresses.

Despite the common elements, treatment is tailored in many ways for hoarding. As illustrated in the case of Dena, many people with hoarding disorder don't recognise the gravity of problems created by their symptoms. Therapy cannot begin to address the hoarding symptoms until the person develops insight. To facilitate insight, motivational strategies are used to help the person consider reasons to change. Once people decide to

change, therapists help them make decisions about their objects and can provide tools and strategies to help them organise and remove their clutter. Therapists often supplement their office sessions with in-home visits, which can help them gain a better sense of the degree of hoarding but also allow for in vivo exercises on de-cluttering. In the first randomised controlled trial of this approach, patients who received CBT demonstrated significantly more improvement than did those assigned to a waiting list (Steketee, Frost, Tolin, Rasmussen, & Brown, 2010). With 26 weeks of treatment, about 70 percent of patients showed at least modest improvement in hoarding symptoms. Self-help groups, supplemented with structured readings, have been found to be a helpful approach that is less expensive than individual therapy (Muroff, Levis, & Bratnietis, 2014).

Early cognitive-behavioural interventions focused on helping clients discard their objects as quickly as possible, hoping to avoid the quagmire of indecision and anxiety that might come from too much focus on evaluating possessions. Unfortunately, patients tended to drop out of treatment and even those who did remain often showed little response (Abramowitz, Franklin, Schwartz, & Furr, 2003; Mataix-Cols, Marks, Greist, Kobak, & Baer, 2002).

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Despite the evidence that removing possessions too rapidly tends to fail, the TV show *Hoarders* featured many people who were faced with ridding themselves of objects under dire threat of eviction or other contingencies and so were forced to discard their collections immediately.



## RETURNING TO CLINICAL CASE

### CBT treatment for Paul

Paul and his therapist agreed to work together using CBT. To begin, they reviewed childhood and current influences on Paul's symptoms. Paul described his parents' extremely high standards for appearance, along with his father's obsessive and perfectionistic style. Throughout his life, Paul had felt that he could not live up to their standards.

He and the therapist then identified several cognitions (thoughts) that seemed to contribute to his anxiety, such as 'Any flaw means I'm ugly' or 'I know my client is thinking about how ugly my nose is'. Paul was asked to record his most negative thoughts each day and was taught ways to evaluate whether these thoughts might be overly harsh. He began to consider alternative ways of thinking.

As Paul developed more positive ways of thinking, the therapist began to target his avoidant and ritualistic behaviours. Paul tended to avoid social events, bright lights and even eye contact with others and he understood that these behaviours were interfering with his life. Treatment consisted of exposure, in which the therapist gradually coached Paul to make eye contact, to engage in social activities and even to talk with others under bright light. Paul also had been conducting a series of rituals to try to reduce his anxiety, including engaging in facial exercises, studying others' noses and surfing plastic surgery websites. The therapist helped him understand that the rituals did not actually relieve his anxiety. Rituals were tackled using response prevention: Paul was asked to stop conducting the rituals and to monitor his mood and anxiety as he did so.

After five sessions of treatment, Paul's therapist introduced perceptual retraining. When people with BDD look in the mirror, they focus on small details of their worst feature and they are overly evaluative. As daily homework, Paul was asked to spend time looking in the mirror but focusing on the whole of his appearance. He was also asked to describe his nose using non-evaluative, objective language. The intense anxiety he initially experienced when completing this exercise diminished within a week. Soon, he was able to appreciate some of his features that he had previously ignored — for example, he noticed that he had nice eyes.

People with body dysmorphic disorder often focus excessive attention on their own appearance. Paul was trained to refocus his attention on people and events outside of himself. For example, when dining with a friend, he was coached to attend to the sound of his friend's voice, the flavour of the food and the content of their conversation.

As Paul made gains, the therapist began to work on the more difficult and core aspects of cognitions — his deeply held beliefs about the meaning of his appearance. Paul described feeling that his physical flaws made him unlovable. The therapist helped him begin to consider his many positive qualities.

The tenth and final session consisted of reviewing the skills he had learned and discussing the strategies he would use if symptoms returned. Over the course of treatment, Paul's symptoms remitted so greatly that by the last session, he was no longer distressed about his nose. (Wilhelm et al., 2010)

#### QUESTIONS

1. What advice would you give Paul for the future?
2. Do you think that there could be a correlation to BDD and rates of plastic surgery? Why or why not?

The family relationships of those with hoarding disorder are often profoundly damaged. Relatives usually try various approaches to help clear the clutter, only to become more and more frustrated and angry as those attempts fail. Many resort to coercive strategies, including removing the hoarder's possessions while the person is away — strategies that typically create mistrust and animosity. Family approaches to hoarding begin by building rapport around these difficult issues (Tompkins & Hartl, 2009). Rather than aiming for a total absence of clutter, family members are urged to identify the aspects of hoarding and clutter that are most dangerous, for example, lack of access to an emergency exit. They can use their concern regarding these issues to begin dialogue and set priorities with the person with hoarding disorder. Support groups and online communities for family members of those with hoarding disorder have become increasingly common.

## 4.8 Post-traumatic stress disorder and acute stress disorder

**LEARNING OUTCOME 4.8** Summarise how the nature and severity of the trauma, as well as biological and psychological risk factors, contribute to whether trauma-related disorders develop.

Post-traumatic stress disorder and acute stress disorder are diagnosed only when a person develops symptoms after a traumatic event. This diagnosis stands in contrast to all other major DSM diagnoses, which are defined entirely by symptom profiles. No other major DSM diagnosis places emphasis squarely on the cause.

## Clinical description and epidemiology of post-traumatic stress disorder and acute stress disorder

**Post-traumatic stress disorder (PTSD)** entails an extreme response to a severe stressor, including recurrent memories of the trauma, avoidance of stimuli associated with the trauma, negative emotions and thoughts, and symptoms of increased arousal. Although people have known for many years that the stresses of combat can have powerful adverse effects on soldiers, the aftermath of the Vietnam War spurred the development of this diagnosis.

The diagnostic criteria define serious trauma as an event that involved actual or threatened death, serious injury or sexual violation. Far too many people have been exposed to these types of traumas. According to Phoenix Australia, 15 million Australians are affected by trauma (Phoenix Australia, n.d.). PTSD support focuses on veterans, victims of assault and those affected by natural disasters such as bushfires.

Exposure to trauma, however, is only the first part of considering this diagnosis. In addition to trauma, the diagnosis of PTSD requires that a set of symptoms be present. In the DSM-5, the symptoms for PTSD are grouped into four major categories.

- *Intrusively re-experiencing the traumatic event.* Like Ashley (see the clinical case below), many people with PTSD have recurrent dreams about the trauma. Others are haunted by painful and intrusive memories, often evoked by small sensory cues that can bring on a wave of psychophysiological arousal. For example, a veteran may tremble when he hears the sound of a helicopter that reminds him of the battlefield; for months after the event, a woman who was raped in a dark room may find her heart pounds when night falls.
- *Avoidance of stimuli associated with the event.* Most people with PTSD strive to avoid thinking about the event and some try to avoid all reminders of it. For example, a Turkish earthquake survivor stopped sleeping indoors after he was buried alive at night (McNally, 2003). Avoidance usually fails: most people say that they do remember the event all too well and often (Rubin, Berntsen, & Bohni, 2008).
- *Other signs of negative mood and thought that developed after the trauma.* Many people with PTSD feel detached from friends and activities, and find that nothing in life brings joy. As they wrestle with questions of blame about the event, many will come to believe that they are bad, and others will develop the belief that all people are untrustworthy.
- *Symptoms of increased arousal and reactivity.* The person with PTSD often feels continuously on guard, monitoring the environment for danger. Laboratory studies have confirmed that people with PTSD demonstrate heightened arousal, as measured by physiological responses to trauma-relevant images (Orr et al., 2003). This can manifest in tendencies to be jumpy when startled, to have outbursts over small events, and to find it challenging to get to sleep or to sleep through the night.

The symptoms of PTSD may develop soon after the trauma but sometimes do not develop for years afterwards. Once PTSD develops, symptoms are relatively chronic. In one study of people diagnosed with PTSD, about half continued to experience diagnosable symptoms when interviewed several years later (Perkonig et al., 2005). Suicidal thoughts are common (Bernal et al., 2007), as are incidents of non-suicidal self-injury (Weierich & Nock, 2008). In a study of 15 288 army veterans followed for

Rescue workers, such as this firefighter who was at the World Trade Center in the aftermath of the 9/11 terrorist attack, could be vulnerable to PTSD.



30 years after their initial military service, PTSD predicted an elevated risk of early death from medical illness, accidents and suicides (Boscarino, 2006).

#### DSM-5

##### DSM-5 criteria for post-traumatic stress disorder

- A. Exposure to actual or threatened death, serious injury or sexual violence, in one or more of the following ways: experiencing the event personally, witnessing the event in person, learning that a violent or accidental death or threat of death occurred to a close other, or experiencing repeated or extreme exposure to aversive details of the event(s) other than through the media.
- B. At least one of the following intrusion symptoms:
  - recurrent, involuntary and intrusive distressing memories of the trauma(s) or in children, repetitive play regarding the trauma themes
  - recurrent distressing dreams related to the event(s)
  - dissociative reactions (e.g., flashbacks) in which the individual feels or acts as if the trauma(s) were recurring or in children, re-enactment of trauma during play
  - intense or prolonged distress or physiological reactivity in response to reminders of the trauma(s).
- C. At least one of the following avoidance symptoms:
  - avoids internal reminders of the trauma(s)
  - avoids external reminders of the trauma(s).
- D. At least two of the following negative alterations in cognitions and mood began after the event:
  - inability to remember an important aspect of the trauma(s)
  - persistent and exaggerated negative beliefs or expectations about one's self, others or the world
  - persistently excessive blame of self or others about the trauma(s)
  - persistently negative emotional state, or in children younger than seven, more frequent negative emotions
  - markedly diminished interest or participation in significant activities
  - feeling of detachment or estrangement from others, or in children younger than seven, social withdrawal
  - persistent inability to experience positive emotions.
- E. At least two of the following changes in arousal and reactivity:
  - irritable or aggressive behaviour
  - reckless or self-destructive behaviour
  - hypervigilance
  - exaggerated startle response
  - problems with concentration
  - sleep disturbance.
- F. The symptoms began or worsened after the trauma(s) and continued for at least one month.
- G. Among children younger than seven, diagnosis requires criteria A, B, E and F, but only one symptom from either category C or D.

It has been argued that prolonged exposure to trauma, such as repeated childhood abuse, might lead to a broader range of symptoms than those covered by the DSM criteria for PTSD. This syndrome has been referred to as complex PTSD (Herman, 1992). Authors differ in the symptom profiles they link to prolonged trauma, but most write about negative emotions, relationship disturbances and negative self-concept (Cloitre et al., 2011). Comprehensive review suggests that prolonged trauma may lead to more severe PTSD symptoms but does not seem to result in a distinct subtype with unique symptomatology (Resick et al., 2012). Given this, the DSM-5 does not include a diagnosis or a specifier for complex PTSD.

In addition to PTSD, the DSM includes a diagnosis for **acute stress disorder (ASD)**. Like PTSD, ASD is diagnosed when symptoms occur after a trauma. The symptoms of ASD are fairly similar to those of PTSD, but the duration is shorter; this diagnosis is only applicable when the symptoms last for three days to one month.

The ASD diagnosis is not as well accepted as the PTSD diagnosis. There are two major concerns about the ASD diagnosis. First, the diagnosis could stigmatise short-term reactions to serious traumas, even though

these are quite common (Harvey & Bryant, 2002). For example, after a rape, as many as 90 percent of women report at least some (subsyndromal) symptoms (Rothbaum, Foa, Murdock, Riggs, & Walsh, 1992). Second, most people who go on to meet diagnostic criteria for PTSD do not experience DSM-IV-TR diagnoses of ASD in the first month after the trauma (Bryant, Creamer, O'Donnell, Silove, & McFarlane, 2008). Nonetheless, those who do experience ASD are at elevated risk of developing PTSD within two years (Bryant, 2011).

### CLINICAL CASE

#### Ashley

Ashley, a 20-year-old university student, sought help at the university counselling centre. Three months before, she had been raped by a man whom she had met at a party that evening. She sought help when the ongoing terror and associated symptoms led her to fail her classes. Since the rape, she had felt as though she was on constant alert, scanning the environment for the man who had assaulted her. When she would see someone who looked even remotely like her attacker, her heart would pound, her knees would shake and she would feel overwhelmed with terror. She had avoided social events because the thought of interacting with men frightened her. Most nights, she had tossed and turned for at least an hour before falling asleep and then intense nightmares of the event would wake her. Her sleep loss often left her too exhausted to attend class and even when she did attend class, she felt distracted and unable to concentrate. As she developed a newly cynical attitude towards people, she had withdrawn from most of her friends. She became unmotivated to be a part of campus life and she stopped attending the club meetings that had been an important part of her social life before the trauma. Although she had tried hard to stop thinking about the event, she faced almost daily reminders, as sexual assaults were a constant issue in the campus newspapers, activist rallies and conversations in the cafeteria.

#### QUESTIONS

1. What DSM-5 criteria for PTSD does Ashley display?
2. Can you think of any local resources that can help those who have been affected by sexual assault?

PTSD tends to be highly comorbid with other conditions. In one study of a representative community sample, researchers conducted diagnostic assessments repeatedly from ages 3 to 26. Among people who had developed PTSD by age 26, almost all (93 percent) had been diagnosed with another psychological disorder before age 21. The most common comorbid disorders are other anxiety disorders, major depression, substance abuse and conduct disorder (Koenen, Moffitt, Poulton, Martin, & Caspi, 2007). Two-thirds of those with PTSD at age 26 had experienced another anxiety disorder by age 21.

Among people exposed to a trauma, women are twice as likely to develop PTSD as are men (Breslau, Chilcoat, Kessler, & Davis, 1999). This finding is consistent with the gender ratio observed for most anxiety disorders. Women may also face different life circumstances than do men. For example, women are much more likely than men to be sexually assaulted during childhood and adulthood (Tolin & Foa, 2006). In studies that control for a history of sexual abuse and assault, men and women have comparable rates of PTSD (Tolin & Foa, 2006).

Culture may shape the risk for PTSD in several ways. Some cultural groups may be exposed to higher rates of trauma and, as a consequence, manifest higher rates of PTSD. Culture also may shape the types of symptoms observed in PTSD. *Ataque de nervios*, originally identified in Puerto Rico, involves physical symptoms and fears of going crazy in the aftermath of severe stress, and thus is similar to PTSD.

## Aetiology of post-traumatic stress disorder

Above, we mentioned that two-thirds of people who develop PTSD have a history of an anxiety disorder. Not surprisingly, then, many of the risk factors for PTSD overlap with the risk factors for anxiety disorders (see table 4.3). For example, PTSD appears to be related to genetic risk for anxiety disorders (Tambs et al., 2009), high levels of activity in areas of the fear circuit such as the amygdala (Rauch et al., 2000), childhood exposure to trauma (Breslau, Davis, & Andreski, 1995) and tendencies to attend selectively

to cues of threat (Bar-Haim et al., 2007). Also parallel with anxiety disorders, neuroticism and negative affectivity predict the onset of PTSD (Pole et al., 2009; Rademaker, van Zuiden, Vermetten, & Geuze, 2011).

As with anxiety disorders, PTSD has been related to Mowrer's two-factor model of conditioning (Keane, Zimering, & Caddell, 1985). In this model, the initial fear in PTSD is assumed to arise from classical conditioning. For example, a man may come to fear walking in the neighbourhood (the conditioned stimulus) where he was assaulted (the unconditioned stimulus). This classically conditioned fear is so intense that the man avoids the neighbourhood as much as possible. Operant conditioning contributes to the maintenance of this avoidance behaviour; the avoidance is reinforced by the reduction of fear that comes from not being in the presence of the conditioned stimulus. This avoidant behaviour interferes with chances for the fear to extinguish.

### **Nature of the trauma: severity and the type of trauma matter**

The severity of the trauma influences whether or not a person will develop PTSD. Consider the case of people who are exposed to war. These effects of PTSD can be intense and long-lasting. In one Australian study on veterans of World War II, '45% of the veterans sampled were found to have active PTSD 45 years after the war' (Kidson, Douglas, & Holwill, 1993).

The residents of New York City after the terrorist attack on the World Trade Center on 11 September 2001 also showed a prevalence of PTSD. Based on a telephone survey after the attack, researchers determined that 7 percent of the adults living within eight miles of the World Trade Center reported symptoms that would have warranted a diagnosis of PTSD, but 20 percent of those living within a mile of the site reported such symptoms (Galea et al., 2002). In short, among people who have been exposed to traumas, those exposed to the most severe traumas are most likely to develop PTSD.

Beyond severity, the nature of the trauma matters. Traumas caused by humans are more likely to cause PTSD than are natural disasters (Charuvastra & Cloitre, 2008). For example, rapes, combat experience, abuse and assault are all associated with higher risk than are natural disasters. Perhaps these events are seen as more distressing because they challenge ideas about humans as benevolent.

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Survivors of natural disasters, such as the Christchurch earthquake in 2011, are at risk for PTSD, but their risk may not be as high as those who experience traumas caused by humans, such as assaults.



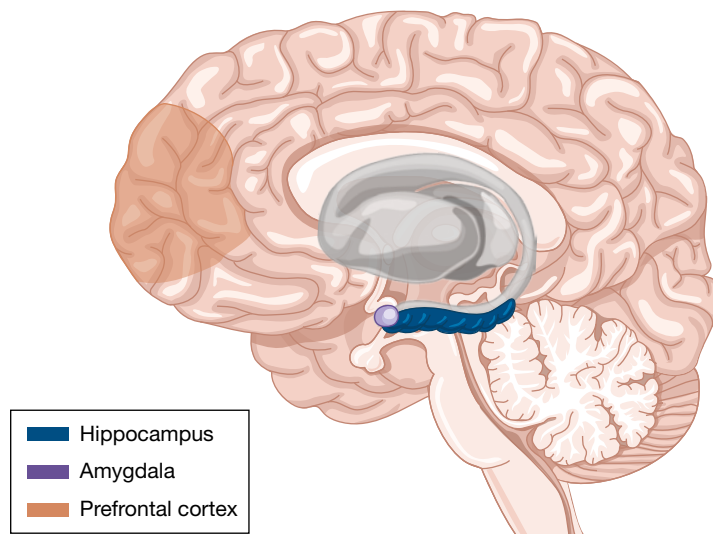
## Neurobiology: the hippocampus

Above, we noted that PTSD appears to be related to dysregulation of the fear circuit, as with anxiety disorders discussed earlier. PTSD appears to be uniquely related to the function of the hippocampus (see figure 4.11) (Shin & Liberzon, 2010). Brain-imaging studies show that the volume of the hippocampus is smaller among people with PTSD than among those who do not have the condition (Bremner et al., 2003). A study of multiple pairs of identical twins, one of whom was a Vietnam veteran and the other not, is revealing of how hippocampal volume and PTSD are related (Gilbertson et al., 2002). As in previous studies of veterans, smaller hippocampal volume was associated with PTSD symptoms, but this study went on to find an additional important pattern. That is, smaller hippocampal volume in the non-veteran twins was related to greater likelihood of PTSD in the veteran twins after military service. This suggests that smaller-than-average hippocampal volume probably precedes the onset of disorder.

The hippocampus plays a central role in our ability to locate autobiographical memories in space, time and context, and in organising our narratives of those memories. Difficulties in organising memories and placing them in context could set the stage for PTSD.

Let's start with the idea of context. People with PTSD experience fear when they have any reminder of their trauma, even out of context. For example, the soldier who witnessed bombs dropping from a plane may remain frightened by all planes after he returns from the war. For those with PTSD, the cues that one is in a safe environment do not seem to work; the fear remains as intense as it did in the moment of the trauma. One argument is that hippocampal deficits could increase the risk that a person will experience this type of fear regardless of whether the context is safe or dangerous (Maren, Phan, & Liberzon, 2013).

**FIGURE 4.11** A smaller hippocampus may be related to the risk of developing PTSD.



Now consider the nature of memory in PTSD. Even though the person has intense memories cued by smells, sounds and other sensory stimuli, he or she often has a hard time organising these memories into well-organised narratives of the event. A meta-analysis of 27 studies found that people diagnosed with PTSD consistently demonstrate deficits on neuropsychological tests of verbal memory even while performing adequately on tests of visual memory (Brewin, Kleiner, Vasterling, & Field, 2007). Brewin (2014) theorises that decreased hippocampal volume could help explain the deficits in verbal memory. Because other regions of the brain are involved in processing memories of physical sensations, the person with PTSD may sustain strong memories for sensory aspects of their trauma. This could help us understand how a person could simultaneously have a hard time describing the trauma and yet still be tortured by reminders of the trauma.

In sum, hippocampal deficits could contribute to psychological vulnerability to PTSD in two different ways. First, the hippocampal deficits could increase the risk that a person will respond to reminders of the trauma even when they occur in safe contexts. Second, the hippocampal deficits may interfere with organising coherent narratives about the trauma.

## Coping

Several types of studies suggest that people who cope with a trauma by trying to avoid thinking about it are more likely than others to develop PTSD. Much of the work on avoidance coping focuses on symptoms of **dissociation** (such as feeling removed from one's body or emotions or being unable to remember the event). We discuss dissociation in more detail in a later chapter. Dissociation may keep the person from confronting memories of the trauma. People who have symptoms of dissociation during and immediately after the trauma are more likely to develop PTSD, as are people who try to suppress memories of the trauma (Ehlers, Mayou, & Bryant, 1998). For example, in one study, researchers interviewed women within two weeks of a rape to ask questions about dissociation during the rape (e.g., 'Did you feel numb?' and 'Did you have moments of losing track of what was going on?'). Women with high levels of dissociation were much more likely to develop PTSD symptoms than were women with low levels of dissociation. The correlation between dissociation and PTSD has been confirmed in a meta-analysis of 16 studies involving 3534 participants (Ozer, Best, Lipsey, & Weiss, 2003). Many studies now show that symptoms of dissociation shortly after being raped predict the development of PTSD (Brewin & Holmes, 2003). Moreover, people who continue to use dissociation in the years after the trauma are at risk for ongoing PTSD symptoms (Briere, Scott, & Weathers, 2005).

To encourage clinicians to consider these coping patterns, the DSM-5 includes a dissociative symptom specifier in order to denote when PTSD is accompanied by persistent or recurrent symptoms of dissociation. About 10–15 percent of people meet the criteria for this specifier (Stein et al., 2013) and it is more common among people who have a history of childhood abuse (Wolf et al., 2012).

Other protective factors may help a person cope with severe traumas more adaptively. Two factors that seem particularly important are high intelligence (Breslau, Lucia, & Alvarado, 2006; Kremen et al., 2007) and strong social support (Brewin, Andrews, & Valentine, 2000). As an example, veterans returning from war who report a stronger sense of social support are less likely to develop PTSD (Vogt et al., 2011). Having better intellectual ability to make sense of horrifying events, and more friends and family members to assist in that process, helps people avoid symptoms after traumatic events.

A surprisingly high proportion of people cope quite well with trauma. For some, trauma awakens an increased appreciation of life, renews a focus on life priorities and provides an opportunity to understand one's strengths in overcoming adversity (Bonanno, 2004; Tedeschi, Park, & Calhoun, 1998).

## 4.9 Treatment of post-traumatic stress disorder and acute stress disorder

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**LEARNING OUTCOME 4.9** Describe the medication and psychological treatments for the trauma-related disorders.

A good deal of work has focused on treatment of PTSD using medication and psychological treatments. Less research is available on ASD.

### Medication treatment of PTSD

Dozens of randomised controlled trials have been conducted to examine medication treatments of PTSD (Stein, Ipser, & Seedat, 2000). One class of antidepressant, the selective serotonin reuptake inhibitors (SSRIs), has received strong support as a treatment. Relapse is common if medications are discontinued.

## Psychological treatment of PTSD

Parallel with the psychological treatment of anxiety disorders discussed earlier, exposure treatment is the primary psychological approach to treating PTSD. In PTSD, the focus of exposure treatment is on memories and reminders of the original trauma. Where possible, the person is directly exposed to reminders of the trauma using **in vivo exposure** — for example, by returning to the scene of the event. In other cases, **imaginal exposure** is used — that is, the person deliberately remembers the event (Keane, Fairbank, Caddell, & Zimering, 1989). Therapists have also used virtual reality technology to treat PTSD; this technology can provide more vivid exposure than some clients may be able to generate in their imaginations (Rothbaum et al., 2006).

Randomised controlled trials suggest that exposure treatment provides more relief from the symptoms of PTSD than supportive unstructured psychotherapy (Powers, Halpern, Ferenschak, Gillihan, & Foa, 2010) or medication (Bradley, Greene, Russ, Dutra, & Westen, 2005). Despite the success of this approach, a great deal of controversy has arisen over another treatment of PTSD and we discuss this controversy in focus on discovery 4.1.

### FOCUS ON DISCOVERY 4.1

#### Eye movement desensitisation and reprocessing

In 1989, Francine Shapiro began to promulgate an approach to trauma treatment called eye movement desensitisation and reprocessing (EMDR). In this procedure, the client recalls a scene related to the trauma. Keeping the scene in mind, the client visually tracks the therapist's fingers as the therapist moves them back and forth about a foot in front of the person's eyes. This process continues for a minute or so or until the client reports that the image is becoming less painful. At this point, the therapist tells the client to say whatever negative thoughts he or she is having, while continuing to track the therapist's fingers. Finally, the therapist tells the client to think a positive thought (e.g., 'I can deal with this') and to hold this thought in mind, still tracking the therapist's fingers. This treatment, then, consists of classic imaginal exposure techniques, along with the extra technique of eye movement.

EMDR proponents argue that the eye movements promote rapid extinction of the conditioned fear and correction of mistaken beliefs about fear-provoking stimuli (Shapiro, 1999). The claims of dramatic efficacy have extended to disorders other than PTSD, including attention-deficit/hyperactivity disorder, dissociative disorders, panic disorder, public-speaking fears, test anxiety and specific phobias (Lohr, Tolin, & Lilienfeld, 1998).

Despite the claims about this approach, several studies have indicated that the eye movement component of treatment is not necessary. Findings from a series of studies indicate that this therapy is no more effective than traditional exposure treatment of PTSD (Seidler & Wagner, 2006). The eye movement component is not supported by adequate theoretical explanations. Gerald Rosen made the following clever comparison. Imagine a practitioner begins to offer exposure treatment wearing a purple hat. We would hardly allow the 'new' approach to merit labelling and marketing as 'purple hat therapy' (Rosen & Davison, 2003). Given the lack of effective innovation in this approach, several researchers have argued that this approach should not be offered (Goldstein, de Beurs, Chambless, & Wilson, 2000).

#### QUESTIONS

1. Do you think EMDR treatment could help clients, even though studies indicate that it is not effective? Explain your answer.
2. What other 'purple hat therapies' have you heard of?

Exposure therapy is hard for both the patient and the therapist because it requires such intense focus on traumatising events. For example, women who have developed PTSD after rape might be asked to vividly recall the details of the worst moments of the attack. The patient's symptoms may even increase temporarily in the initial stages of therapy (Keane, Gerardi, Quinn, & Litz, 1992).

Treatment is likely to be particularly hard and to require more time when the client has experienced recurrent traumas during childhood, as those experiences can interfere with learning to cope with emotions. In one randomised controlled trial, researchers examined whether teaching skills to regulate emotions would bolster the effects of exposure treatment for women who reported PTSD symptoms as a consequence of childhood abuse. The addition of emotion regulation skills led to several positive gains compared to standard exposure treatment, including diminished PTSD symptoms, improved emotion regulation, enhanced interpersonal functioning and lower rates of post-treatment symptom relapse (Cloitre et al., 2010).

Several cognitive strategies have been used to supplement exposure treatment for PTSD. Interventions designed to bolster people's beliefs in their ability to cope with the initial trauma have been shown to fare well in a series of studies (Keane, Marshall, & Taft, 2006). Cognitive processing therapy is designed to help victims of rape and childhood sexual abuse dispute tendencies towards self-blame. This approach has also received empirical support (Chard, 2005; Resick, Nishith, Weaver, Astin, & Feuer, 2002) and appears to be particularly helpful in reducing guilt (Resick, Nishith, & Griffin, 2003) and dissociation (Resick, Suvak, Johnides, Mitchell, & Iverson, 2012).

#### FOCUS ON DISCOVERY 4.2

##### Critical incident stress debriefing

Critical incident stress debriefing (CISD) involves immediate treatment of trauma victims within 72 hours of the traumatic event (Mitchell & Everly, 2000). Unlike cognitive-behavioural treatment, CISD is usually limited to one long session and is given regardless of whether the person has developed symptoms. Therapists encourage people to remember the details of the trauma and to express their feelings as fully as they can. Therapists who practise this approach often visit disaster sites immediately after events, sometimes invited by local authorities (as in the aftermath of the World Trade Center attack) and sometimes not; they offer therapy both to victims and to their families.

Like EMDR, CISD is highly controversial. A review of six studies, all of which included randomly assigning clients to receive CISD or no treatment, found that those who received CISD tended to fare worse (Litz, Gray, Bryant, & Adler, 2002). No one is certain why harmful effects occur, but remember that many people who experience a trauma do not develop PTSD. Many experts are dubious about the idea of providing therapy for people who have not developed a disorder. Some researchers raise the objection to CISD that a person's natural coping strategies may work better than those recommended by someone else (Bonanno et al., 2002).

##### QUESTIONS

1. Do you think it's best to provide a 'preventive' approach such as CISD to those who might be susceptible to PTSD, but are currently not showing symptoms? Why or why not?
2. What do you believe is the rationale for revisiting the disaster sites shortly after the traumatic event? Do you think there is some merit to this?

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## SUMMARY

### **4.1 Describe the clinical features of the anxiety disorders.**

As a group, anxiety disorders are the most common type of psychological disorder. Specific phobia is defined by an intense fear of an object or situation, social anxiety disorder by an intense fear of strangers or social scrutiny, panic disorder by anxiety about recurrent panic attacks, agoraphobia by a fear of places where escaping or getting help would be difficult if anxiety symptoms were to occur, and generalised anxiety disorder by worries lasting at least six months.

### **4.2 Describe how the anxiety disorders tend to co-occur and understand how gender and culture influence the prevalence of anxiety disorders.**

People with one anxiety disorder are very likely to experience a second anxiety disorder during their life. About 60 percent of people with anxiety disorders will experience major depression during their life. Women are much more likely than men to report an anxiety disorder. Culture influences the focus of fears, the ways that symptoms are expressed and even the prevalence of different anxiety disorders.

### **4.3 Recognise commonalities in aetiology across the anxiety disorders, as well as the factors that shape the expression of specific anxiety disorders.**

Many risk factors set the stage for anxiety disorders generally, rather than for a specific anxiety disorder. Behavioural models of anxiety disorders build from Mowrer's two-factor model (classical conditioning followed by operant conditioning). These models have been extended to consider that the classical conditioning may be driven by direct exposure to an event, observation of someone else experiencing an event (modelling) or verbal instruction. Other risk factors may increase the propensity towards fear conditioning. Genes increase risk for anxiety disorders. Neurobiological research on anxiety focuses on the brain's fear circuit. Anxiety disorders also appear to involve poor functioning of the gamma-aminobutyric acid (GABA), norepinephrine and serotonin systems. The personality traits of behavioural inhibition and neuroticism are both related to the development of anxiety disorders. From the cognitive perspective, anxiety disorders are associated with negative expectations about the future, beliefs that life is uncontrollable and a bias to attend to negative information.

Specific phobias are believed to reflect conditioning in response to a traumatic event. Prepared learning refers to the fact that people are likely to sustain conditioned responses to fear stimuli that have some evolutionary significance.

Social anxiety disorder appears to be related to conditioning and behavioural inhibition. Cognitive factors involved in social anxiety disorder include self-critical evaluations of social performance and tendencies to focus on internal thoughts and sensations.

A cognitive model of agoraphobia focuses on 'fear of fear', or overly negative beliefs about what will happen if one experiences anxiety.

One cognitive model emphasises that, with generalised anxiety disorder (GAD), worry might actually protect people from intensely disturbing emotional images. People with GAD also appear to be intolerant of ambiguity.

### **4.4 Describe treatment approaches that are common across the anxiety disorders and how treatment approaches are modified for the specific anxiety disorders.**

Exposure treatment is the most validated psychological treatment for anxiety disorders. Cognitive treatments supplement exposure with interventions to challenge negative beliefs about what will happen when a person faces his or her fears.

For specific phobias, exposure treatments can work quite quickly. For social anxiety disorder, cognitive strategies, such as teaching a person to focus less on internal thoughts and sensations, are a helpful addition to exposure treatment. The most effective treatments for panic disorder include exposure to somatic sensations, along with cognitive techniques to challenge catastrophic

misinterpretations of those symptoms. Cognitive-behavioural treatment of GAD can include relaxation training, strategies to help a person tolerate uncertainty and face core fears, and specific tools to combat tendencies to worry.

#### **4.5 Define the symptoms and epidemiology of the obsessive-compulsive and related disorders and the trauma-related disorders.**

The major obsessive-compulsive and related disorders include obsessive-compulsive disorder (OCD), body dysmorphic disorder (BDD) and hoarding disorder. OCD is defined by obsessions and/or compulsions. BDD is defined by a preoccupation with an imagined defect in appearance and by intensive behavioural attempts to cope with the imagined defect. Hoarding disorder is defined by excessive acquisition of objects and severe difficulties in discarding objects, even when they are objectively without value.

People with BDD and hoarding disorder often have a history of OCD. Beyond this, the obsessive-compulsive and related disorders are commonly comorbid with anxiety disorders and major depressive disorder. OCD and BDD are often comorbid with substance abuse.

#### **4.6 Describe the commonalities in the aetiology of obsessive-compulsive and related disorders, as well as the factors that shape the expression of the specific disorders within this cluster.**

OCD, BDD and hoarding disorder are moderately heritable. People with BDD and hoarding disorder often report a family history of OCD. OCD is characterised by high activity in the orbitofrontal cortex, the caudate nucleus and the anterior cingulate. BDD involves heightened activity of the orbitofrontal cortex and the caudate nucleus. Hoarding disorder is related to heightened activity in the orbitofrontal cortex and the anterior cingulate.

Cognitive-behavioural models help explain why one might develop a specific disorder in the cluster of obsessive-compulsive and related disorders. A behavioural model of OCD suggests that people sustain conditioned responses long after the contingencies that conditioned the initial behaviour have shifted. Psychologically, OCD appears to be characterised by a deficit in yedasentience. Thought suppression may exacerbate tendencies towards obsessions. The cognitive model of BDD focuses on a detail-oriented analytic style, tendencies to overvalue the meaning of appearance for self-worth and excessive attention to cues related to appearance. Cognitive-behavioural models of hoarding disorder focus on poor organisational abilities (difficulties with attention, categorisation and decision making), unusual beliefs about possessions and avoidance behaviours.

#### **4.7 Describe the medication and psychological treatments for the obsessive-compulsive and related disorders.**

Antidepressants are the most supported medication treatment for the obsessive-compulsive and related disorders. The major psychological treatment approach for the obsessive-compulsive and related disorders is exposure and response prevention (ERP). ERP for hoarding disorder often involves motivational strategies to enhance insight and willingness to consider change.

#### **4.8 Summarise how the nature and severity of the trauma, as well as biological and psychological risk factors, contribute to whether trauma-related disorders develop.**

Post-traumatic stress disorder (PTSD) and acute stress disorder (ASD) are both severe reactions to trauma. The symptoms of PTSD and ASD are similar, but ASD can only be diagnosed if symptoms have lasted less than one month. Most people who develop PTSD have a history of other psychological disorders and two-thirds have a history of anxiety disorders.

Some of the general risk factors for anxiety disorders appear to apply to PTSD: genetic vulnerability, amygdala hyperactivity, neuroticism, childhood trauma exposure and tendencies to attend to cues of threat in the environment. PTSD has also been related to Mowrer's two-factor model of conditioning.

More specific risk factors for PTSD have also been identified. The likelihood that a person will develop PTSD depends on the severity of the trauma. Neurobiological research has found that

people with small hippocampal volume are more likely to develop PTSD. After exposure to trauma, people who rely on dissociative coping strategies (i.e., who avoid thinking about the trauma) are more likely to develop PTSD than people who rely on other strategies. Other resources that might promote adaptive coping, such as higher IQ and stronger social support, can protect against the development of PTSD.

#### 4.9 Describe the medication and psychological treatments for the trauma-related disorders.

Selective serotonin reuptake inhibitors (SSRIs) have been shown in numerous controlled trials to be an effective means of treatment for post-traumatic stress disorder. As with the treatment of anxiety disorders, exposure treatment is the primary psychological approach to treating PTSD, where the focus is on memories and reminders of the original trauma. Treatment is particularly challenging when the client has experienced recurrent traumas during childhood. Cognitive processing therapy is designed to help victims of rape and childhood sexual abuse dispute tendencies towards self-blame, and has been received empirical support.

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## KEY TERMS

**acute stress disorder** a short-lived anxiety reaction to a traumatic event; if it lasts more than a month, it is diagnosed as post-traumatic stress disorder

**agoraphobia** literally, fear of the marketplace; anxiety disorder in which the person fears situations in which it would be embarrassing or difficult to escape if panic symptoms occurred; most commonly diagnosed in some individuals with panic disorder

**anxiety** an unpleasant feeling of fear and apprehension accompanied by increased physiological arousal; can be assessed by self-report, measuring physiological arousal and observing overt behaviour

**anxiety disorders** disorders in which fear or anxiety is overriding; include phobic disorders, social anxiety disorder, panic disorder, generalised anxiety disorder and agoraphobia

**anxiolytics** minor tranquilisers or benzodiazepines used to treat anxiety disorders

**behavioural inhibition** the tendency to exhibit anxiety or to freeze when facing threat. In infants, it manifests as a tendency to become agitated and cry when faced with novel stimuli and may be a heritable predisposition for the development of anxiety disorders

**benzodiazepines** any of several drugs commonly used to treat anxiety, such as Valium and Xanax

**body dysmorphic disorder (BDD)** a disorder marked by preoccupation with an imagined or exaggerated defect in appearance — for example, facial wrinkles or excess facial or body hair

**caudate nucleus** a nucleus within the basal ganglia involved in learning and memory that is implicated in body dysmorphic disorder and obsessive-compulsive disorder

**compulsion** the irresistible impulse to repeat an irrational act or thought over and over again

**depersonalisation** an alteration in perception of the self in which the individual loses a sense of reality and feels estranged from the self and perhaps separated from the body; may be a temporary reaction to stress and fatigue or part of panic disorder, depersonalisation disorder or schizophrenia

**derealisation** loss of the sense that the surroundings are real; present in several psychological disorders, such as panic disorder, depersonalisation disorder and schizophrenia

**dissociation** a process whereby a group of mental processes is split off from the main stream of consciousness or behaviour loses its relationship with the rest of the personality

**exposure and response prevention (ERP)** the most widely used and accepted treatment of obsessive-compulsive disorder, in which the sufferer is prevented from engaging in compulsive ritual activity and instead faces the anxiety provoked by the stimulus, leading eventually to extinction of the conditioned response (anxiety)

**fear** a reaction to real or perceived immediate danger in the present; can involve arousal or sympathetic nervous system activity

**fear circuit** set of brain structures, including the amygdala, that tend to be activated when the individual is feeling anxious or fearful; especially active among people with anxiety disorders

**fear-of-fear hypothesis** a cognitive model for the aetiology of agoraphobia; suggests the condition is driven by negative thoughts about the consequences of having a panic attack in public

**generalised anxiety disorder (GAD)** a disorder characterised by chronic, persistent anxiety and worry

**hoarding disorder** a disorder in which the person has a compulsive need to acquire objects and extreme difficulty in disposing of those objects

**imaginal exposure** treatment for anxiety disorders that involves visualising feared scenes for extended periods of time; frequently used in the treatment of post-traumatic stress disorder when in vivo exposure to the initial trauma cannot be conducted

**in vivo exposure** real-life exposure to the object or situation that is feared, such as exposure to a phobia (such as a snake) or returning to the location where the traumatic event has taken place

**interoceptive conditioning** classical conditioning of panic attacks in response to internal bodily sensations of arousal (as opposed to the external situations that trigger anxiety)

**locus coeruleus** the brain region in the fear circuit that is especially important in panic disorder; the major source in the brain of norepinephrine, which helps trigger sympathetic nervous system activity

**medial prefrontal cortex** a region of the cortex in the anterior frontal lobes involved in executive function and emotion regulation that is implicated in mood and anxiety disorders

**Mowrer's two-factor model** Mowrer's theory of avoidance learning according to which (1) fear is attached to a neutral stimulus by pairing it with a noxious unconditioned stimulus and (2) a person learns to escape the fear elicited by the conditioned stimulus, thereby avoiding the unconditioned stimulus

**obsession** an intrusive and recurring thought that seems irrational and uncontrollable to the person experiencing it

**obsessive-compulsive disorder (OCD)** an anxiety disorder in which the mind is flooded with persistent and uncontrollable thoughts or the individual is compelled to repeat certain acts again and again, causing significant distress and interference with everyday functioning

**orbitofrontal cortex** the portion of the frontal lobe located just above the eyes; one of three closely related brain regions that are unusually active in individuals with obsessive-compulsive disorder

**panic attack** a sudden attack of intense apprehension, terror and impending doom, accompanied by symptoms such as laboured breathing, nausea, chest pain, feelings of choking and smothering, heart palpitations, dizziness, sweating and trembling

**panic disorder** an anxiety disorder in which the individual has sudden, inexplicable and frequent panic attacks

**post-traumatic stress disorder (PTSD)** an extreme response to a severe stressor, including recurrent memories of the trauma, avoidance of associated stimuli, negative emotions and thoughts, and symptoms of increased arousal

**prepared learning** in classical conditioning theory, a biological predisposition to associate particular stimuli readily with the unconditioned stimulus

**safety behaviours** behaviours used to avoid experiencing anxiety in feared situations, such as the tendency of people with social phobia to avoid looking at other people (so as to avoid perceiving negative feedback) or the tendency of people with panic disorder to avoid exercise (so as to avoid somatic arousal that could trigger a panic attack)

**serotonin-norepinephrine reuptake inhibitors (SNRIs)** any of various drugs that inhibit the presynaptic reuptake of serotonin and norepinephrine, such that both neurotransmitters will have more prolonged effects on postsynaptic neurons

**social anxiety disorder (social phobia)** a collection of fears linked to the presence of other people

**specific phobia** an unwarranted fear and avoidance of a specific object or circumstance, for example, fear of non-poisonous snakes or fear of heights

**thought suppression** key feature of obsessive-compulsive disorder; has the paradoxical effect of inducing preoccupation with the object of thought

**yedasentience** the subjective sense of knowing that one has achieved closure on an action or thought; theorised to be deficient among persons with obsessive-compulsive disorder

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## WEBSITES

1. SANE Australia provides resources and information related to mental illness. ([www.sane.org](http://www.sane.org))
  2. Phoenix Australia is a centre for post-traumatic mental health in Australia. (<http://phoenixaustralia.org>)
  3. OCD Stop! Program is a free self-help program for people with OCD. ([www.swinburne.edu.au/lss/bpsyc/clinical-and-health-research/ocd](http://www.swinburne.edu.au/lss/bpsyc/clinical-and-health-research/ocd))
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## REFERENCES

- Abramowitz, J. S., Franklin, M. E., Schwartz, S. A., & Furr, J. M. (2003). Symptom presentation and outcome of cognitive-behavioral therapy for obsessive-compulsive disorder. *Journal of Consulting and Clinical Psychology, 71*, 1049–1057.
- Acarturk, C., de Graaf, R., van Straten, A., Have, M. T., & Cuijpers, P. (2008). Social phobia and number of social fears, and their association with comorbidity, health-related quality of life and help seeking: A population-based study. *Social Psychiatry and Psychiatric Epidemiology, 43*, 273–279.
- Alonso, J., Petukhova, M., Vilagut, G., Chatterji, S., Heeringa, S., Ustun, T. B., et al. (2011). Days out of role due to common physical and mental conditions: Results from the WHO World Mental Health surveys. *Molecular Psychiatry, 16*, 1234–1246.
- Amir, N., Beard, C., Burns, M., & Bomyea, J. (2009). Attention modification program in individuals with generalized anxiety disorder. *Journal of Abnormal Psychology, 118*, 28–33.
- Amir, N., Cashman, L., & Foa, E. B. (1997). Strategies of thought control in obsessive-compulsive disorder. *Behaviour Research and Therapy, 35*, 775–777.
- Andrews, G., Charney, D. S., Sirovatka, P. J., & Regier, D. A. (Eds.). (2009). *Stress-induced and fear circuitry disorders*. Arlington, VA: American Psychiatric Association.
- Ashbaugh, A. R., Antony, M. M., McCabe, R. E., Schmidt, L. A., & Swinson, R. P. (2005). Self-evaluative biases in social anxiety. *Cognitive Therapy and Research, 29*, 387–398.
- Asmundson, G. J., Larsen, D. K., & Stein, M. B. (1998). Panic disorder and vestibular disturbance: An overview of empirical findings and clinical implications. *Journal of Psychosomatic Research, 44*, 107–120.
- Australian Bureau of Statistics (ABS). (2008). *National survey of mental health and wellbeing: summary of results, 2007*, cat. no. 4326.0. Retrieved from [www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/6AE6DA447F985FC2CA2574EA00122BD6/\\$File/43260\\_2007.pdf](http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/6AE6DA447F985FC2CA2574EA00122BD6/$File/43260_2007.pdf)
- Bandelow, B., Zohar, J., Hollander, E., Kasper, S., Möller, H. J., & WFSBP Task Force on Treatment Guide. (2008). World Federation of Societies of Biological Psychiatry (WFSBP) Guidelines for the pharmacological treatment of anxiety, obsessive-compulsive and post-traumatic stress disorders—First Revision. *World Journal of Biological Psychiatry, 9*, 248–312.
- Bar-Haim, Y., Lamy, D., Pergamin, L., Bakermans-Kranenburg, M. J., & van, I. M. H. (2007). Threat-related attentional bias in anxious and nonanxious individuals: A meta-analytic study. *Psychological Bulletin, 133*, 1–24.
- Barlow, D. H. (2004). *Anxiety and its disorders: The nature and treatment of anxiety and panic*. New York: Guilford Press.
- Barlow, D. H., Blanchard, E. B., Vermilyea, J. A., Vermilyea, B. B., & DiNardo, P. A. (1986). Generalized anxiety and generalized anxiety disorder: Description and reconceptualization. *American Journal of Psychiatry, 143*, 40–44.
- Bates, G. W., Campbell, I. M., & Burgess, P. M. (1990). Assessment of articulated thoughts in social anxiety: Modification of the ATSS procedure. *British Journal of Clinical Psychology, 29*, 91–98.
- Belluck, P. (2014, June 15). Thinking of ways to harm her: New findings on timing and range of maternal mental illness. *New York Times*.
- Bernal, M., Haro, J. M., Bernert, S., Brugha, T., de Graaf, R., Bruffaerts, R., et al. (2007). Risk factors for suicidality in Europe: Results from the ESEMED study. *Journal of Affective Disorders, 101*, 27–34.
- beyondblue. (n.d.). *Statistics and references*. Retrieved from [www.beyondblue.org.au/about-us/research-projects/statistics-and-references?sec=sec-anx](http://www.beyondblue.org.au/about-us/research-projects/statistics-and-references?sec=sec-anx)
- Blair, K. S., Shaywitz, J., Smith, B. W., Rhodes, R., Geraci, M., Jones, M., et al. (2008). Response to emotional expressions in generalized social phobia and generalized anxiety disorder: Evidence for separate disorders. *American Journal of Psychiatry, 165*, 1193–1202.
-

- Bloch, M. H., Landeros-Weisenberger, A., Sen, S., Dombrowski, P., Kelmendi, B., Coric, V., et al. (2008). Association of the serotonin transporter polymorphism and obsessive-compulsive disorder: Systematic review. *American Journal of Medical Genetics Part B, Neuropsychiatric Genetics*, 147B, 850–858.
- Bohne, A., Keuthen, N. J., Wilhelm, S., Deckersback, T., & Jenike, M. A. (2002). Prevalence of symptoms of body dysmorphic disorder and its correlates: A cross-cultural comparison. *Psychosomatics*, 43, 486–490.
- Bonanno, G. A. (2004). Loss, trauma, and human resilience: Have we underestimated the human capacity to thrive after extremely aversive events? *American Psychologist*, 59, 20–28.
- Bonanno, G. A., Wortman, C. B., Lehman, D. R., Tweed, R. G., Haring, M., Sonnega, J., et al. (2002). Resilience to loss and chronic grief: A prospective study from preloss to 18-months postloss. *Journal of Personality and Social Psychology*, 83, 1150–1164.
- Borkovec, T. D., & Newman, M. G. (1998). Worry and generalized anxiety disorder. In P. Salkovskis (Ed.), *Comprehensive clinical psychology* (Vol. 6, 439–459). Oxford, UK: Elsevier.
- Boscarino, J. A. (2006). Posttraumatic stress disorder and mortality among U.S. Army veterans 30 years after military service. *Annals of Epidemiology*, 16, 248–256.
- Bouton, M. E., & Waddell, J. (2007). Some biobehavioral insights into persistent effects of emotional trauma. In L. J. Kirmayer, R. Lemelson & M. Barad (Eds.), *Understanding trauma: Integrating biological, clinical, and cultural perspectives* (pp. 41–59). New York: Cambridge University Press.
- Bouton, M. E., Mineka, S., & Barlow, D. H. (2001). A modern learning theory perspective on the etiology of panic disorder. *Psychological Review*, 108, 4–32.
- Bradley, R., Greene, J., Russ, E., Dutra, L., & Westen, D. (2005). A multidimensional meta-analysis of psychotherapy for PTSD. *American Journal of Psychiatry*, 162, 214–227.
- Brakoulias, V., Starcevic, V., Sammut, P., Berle, D., Milicevic, D., Moses, K., et al. (2011). Obsessive-compulsive spectrum disorders: A comorbidity and family history perspective. *Australasian Psychiatry*, 19, 151–155.
- Bremner, J. D., Vythilingam, M., Vermetten, E., Southwick, S. M., McGlashan, T., Nazeer, A., et al. (2003). MRI and PET study of deficits in hippocampal structure and function in women with childhood sexual abuse and posttraumatic stress disorder. *American Journal of Psychiatry*, 160, 924–932.
- Breslau, N., Chilcoat, H. D., Kessler, R. C., & Davis, G. C. (1999). Previous exposure to trauma and PTSD effects of subsequent trauma: Results from the Detroit Area Survey of Trauma. *American Journal of Psychiatry*, 156, 902–907.
- Breslau, N., Davis, G. C., & Andreski, P. (1995). Risk factors for PTSD-related traumatic events: A prospective analysis. *American Journal of Psychiatry*, 152, 529–535.
- Breslau, N., Lucia, V., & Alvarado, G. F. (2006). Intelligence and other predisposing factors in exposure to trauma and posttraumatic stress disorder: A follow-up study at age 17 years. *Archives of General Psychiatry*, 63, 1238–1245.
- Brewin, C. R. (2014). Episodic memory, perceptual memory, and their interaction: Foundations for a theory of posttraumatic stress disorder. *Psychological Bulletin*, 140, 69–97.
- Brewin, C. R., & Holmes, E. A. (2003). Psychological theories of posttraumatic stress disorder. *Clinical Psychology Review*, 23, 339–376.
- Brewin, C. R., Andrews, B., & Valentine, J. D. (2000). Meta-analysis of risk factors for posttraumatic stress disorder in trauma-exposed adults. *Journal of Consulting and Clinical Psychology*, 68, 748–766.
- Brewin, C. R., Kleiner, J. S., Vasterling, J. J., & Field, A. P. (2007). Memory for emotionally neutral information in posttraumatic stress disorder: A meta-analytic investigation. *Journal of Abnormal Psychology*, 116, 448–463.
- Briere, J., Scott, C., & Weathers, F. (2005). Peritraumatic and persistent dissociation in the presumed etiology of PTSD. *American Journal of Psychiatry*, 162, 2295–2301.
- Britton, J., Grillon, C., Lissek, S., Norcross, M. A., Szuhany, K. L., Chen, G., et al. (2013). Response to learned threat: An fMRI study in adolescent and adult anxiety. *American Journal of Psychiatry*, 170.
- Brown, T. A. (2007). Temporal course and structural relationships among dimensions of temperament and DSM-IV anxiety and mood disorder constructs. *Journal of Abnormal Psychology*, 116, 313–328.
- Brown, T. A., Campbell, L. A., Lehman, C. L., Grisham, J. R., & Mancill, R. B. (2001). Current and lifetime comorbidity of the DSM-IV anxiety and mood disorders in a large clinical sample. *Journal of Abnormal Psychology*, 110, 585–599.
- Bryant, R. A. (2011). Acute stress disorder as a predictor of posttraumatic stress disorder: A systematic review. *Journal of Clinical Psychiatry*, 72, 233–239.
- Bryant, R. A., Creamer, M., O'Donnell, M. L., Silove, D., & McFarlane, A. C. (2008). A multisite study of the capacity of acute stress disorder diagnosis to predict posttraumatic stress disorder. *Journal of Clinical Psychiatry*, 69, 923–929.
- Buhlmann, U., Glaesmer, H., Mewes, R., Fama, J. M., Wilhelm, S., Brähler, E., et al. (2010). Updates on the prevalence of body dysmorphic disorder: A population-based survey. *Psychiatry Research*, 178, 171–175.
- Chaffin, M., Silovsky, J. F., & Vaughn, C. (2005). Temporal concordance of anxiety disorders and child sexual abuse: implications for direct versus artifactual effects of sexual abuse. *Journal of Clinical Child and Adolescent Psychology*, 34, 210–222.
- Chambless, D. L., Caputo, G. C., Bright, P., & Gallagher, R. (1984). Assessment of fear of fear in agoraphobics: The body sensations questionnaire and the agoraphobic cognitions questionnaire. *Journal of Consulting and Clinical Psychology*, 52, 1090–1097.
- Chard, K. M. (2005). An evaluation of cognitive processing therapy for the treatment of posttraumatic stress disorder related to childhood sexual abuse. *Journal of Consulting and Clinical Psychology*, 73, 965–971.

- Charuvastra, A., & Cloitre, M. (2008). Social bonds and posttraumatic stress disorder. *Annual Review of Psychology*, 59, 301–328.
- Chavira, D. A., Stein, M. B., & Malcarne, V. L. (2002). Scrutinizing the relationship between shyness and social phobia. *Journal of Anxiety Disorders*, 16, 585–598.
- Chorpita, B. F., Brown, T. A., & Barlow, D. H. (1998). Perceived control as a mediator of family environment in etiological models of childhood anxiety. *Behavior Therapy*, 29, 457–476.
- Chronis-Tuscano, A., Degnan, K. A., Pine, D. S., Perez-Edgar, K., Henderson, H. A., Diaz, Y., et al. (2009). Stable early maternal report of behavioral inhibition predicts lifetime social anxiety disorder in adolescence. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48, 928–935.
- Cisler, J. M., & Koster, E. H. (2010). Mechanisms of attentional biases towards threat in anxiety disorders: An integrative review. *Clinical Psychology Review*, 30, 203–216.
- Clark, D. A. (2006). *Cognitive-Behavioral Therapy for OCD*. New York: Guilford Press.
- Clark, D. M. (1996). Panic disorder: From theory to therapy. In P. M. Salkovskis (Ed.), *Frontiers of Cognitive Therapy* (pp. 318–344). New York: Guilford Press.
- Clark, D. M. (1997). Panic disorder and social phobia. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behaviour Therapy* (pp. 121–153). Oxford, UK: Oxford University Press.
- Clark, D. M., & Wells, A. (1995). A cognitive model of social phobia. In R. Heimberg, M. R. Liebowitz, D. A. Hope & F. R. Schneier (Eds.), *Social phobia: Diagnosis, assessment and treatment* (pp. 69–93). New York: Guilford Press.
- Clark, D. M., Salkovskis, P. M., Hackmann, A., Wells, A., Ludgate, J., & Gelder, M. (1999). Brief cognitive therapy for panic disorder: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 67, 583–589.
- Cloitre, M., Courtois, C. A., Charuvastra, A., Carapezza, R., Stolbach, B. C., & Green, B. L. (2011). Treatment of complex PTSD: Results of the ISTSS expert clinician survey on best practices. *Journal of Traumatic Stress*, 24, 615–627.
- Cloitre, M., Stovall-McClough, K. C., Nooner, K., Zorbas, P., Cherry, S., Jackson, C. L., et al. (2010). Treatment for PTSD related to childhood abuse: A randomized controlled trial. *American Journal of Psychiatry*, 167, 915–924.
- Conceicao do Rosario-Campos, M., Leckman, J. F., Mercadante, M. T., Shavitt, R. G., Prado, H. S., Sada, P., et al. (2001). Adults with early-onset obsessive-compulsive disorder. *American Journal of Psychiatry*, 158, 1899–1903.
- Cook, M., & Mineka, S. (1989). Observational conditioning of fear to fear-relevant versus fear-irrelevant stimuli in rhesus monkeys. *Journal of Abnormal Psychology*, 98, 448–459.
- Cox, B. J., Clara, I. P., & Enns, M. W. (2002). Posttraumatic stress disorder and the structure of common mental disorders. *Depression and Anxiety*, 15, 168–171.
- Craske, M. G., & Mystkowski, J. (2006). Exposure therapy and extinction: Clinical studies. In M. G. Craske, D. Hermans & D. Vansteenwegen (Eds.), *Fear and learning from basic processes to clinical implications* (pp. 217–233). Washington DC: American Psychological Association.
- Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008). Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, 46, 5–27.
- Craske, M. G., Rauch, S. L., Ursano, R., Prenoveau, J., Pine, D. S., & Zinbarg, R. E. (2009). What is an anxiety disorder? *Depression and Anxiety*, 26, 1066–1085.
- Cuijpers, P., Sijbrandij, M., Koole, S. L., Andersson, G., Beekman, A. T., & Reynolds, C. F. (2013). The efficacy of psychotherapy and pharmacotherapy in treating depressive and anxiety disorders: a meta-analysis of direct comparisons. *World Psychiatry*, 12, 137–148.
- Dandeneau, S. D., Baldwin, M. W., Baccus, J. R., Sakellaropoulou, M., & Pruessner, J. C. (2007). Cutting stress off at the pass: Reducing vigilance and responsiveness to social threat by manipulating attention. *Journal of Personality and Social Psychology*, 93, 651–666.
- Dawson, M. E., Schell, A. M., & Banis, H. T. (1986). Greater resistance to extinction of electrodermal responses conditioned to potentially phobic CSs: A noncognitive process? *Psychophysiology*, 23, 552–561.
- de Graaf, R., Bijl, R. V., Ravelli, A., Smit, F., & Vollenbergh, W. A. M. (2002). Predictors of first incidence of DSM-III-R psychiatric disorders in the general population: Findings from the Netherlands mental health survey and incidence study. *Acta Psychiatrica Scandinavica*, 106, 303–313.
- Deacon, B. J., & Abramowitz, J. S. (2004). Cognitive and behavioral treatments for anxiety disorders: A review of meta-analytic findings. *Journal of Clinical Psychology*, 60, 429–441.
- DeRubeis, R. J., & Crits-Christoph, P. (1998). Empirically supported individual and group psychological treatments for adult mental disorders. *Journal of Consulting and Clinical Psychology*, 66, 37–52.
- Didie, E. R., Menard, W., Stern, A. P., & Phillips, K. A. (2008). Occupational functioning and impairment in adults with body dysmorphic disorder. *Comprehensive Psychiatry*, 49, 561–569.
- Domschke, K., Stevens, S., Pfleiderer, B., & Gerlach, A. L. (2010). Interoceptive sensitivity in anxiety and anxiety disorders: An overview and integration of neurobiological findings. *Clinical Psychology Review*, 30, 1–11.
- Dugas, M. J., Marchand, A., & Ladouceur, R. (2005). Further validation of a cognitive-behavioral model of generalized anxiety disorder: Diagnostic and symptom specificity. *Journal of Anxiety Disorders*, 19, 329–343.
- Ehlers, A., Mayou, R. A., & Bryant, B. (1998). Psychological predictors of chronic posttraumatic stress disorder after motor vehicle accidents. *Journal of Abnormal Psychology*, 107, 508–519.

- Eisen, J. L., Sibrava, N. J., Boisseau, C. L., Mancebo, M. C., Stout, R. L., Pinto, A., et al. (2013). Five-year course of obsessive-compulsive disorder: Predictors of remission and relapse. *Journal of Clinical Psychiatry*, 74, 233–239.
- Feusner, J. D., Phillips, K. A., & Stein, D. J. (2010). Olfactory reference syndrome: Issues for DSM-V. *Depression and Anxiety*, 27, 592–599.
- Finlay-Jones, R. (1989). Anxiety. In G. W. Brown & T. O. Harris (Eds.), *Life events and illness* (pp. 95–112). New York: Guilford Press.
- Foa, E. B., & Franklin, M. E. (2001). Obsessive-compulsive disorder. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders* (pp. 209–263). New York: Guilford Press.
- Foa, E. B., & Meadows, E. A. (1997). Psychosocial treatments for posttraumatic stress disorder: A critical review. *Annual Review of Psychology*, 48, 449–480.
- Foa, E. B., Libowitz, M. R., Kozak, M. J., Davies, S., Campeas, R., Franklin, M. E., et al. (2005). Randomized, placebo-controlled trial of exposure and ritual prevention, clomipramine, and their combination in the treatment of obsessive-compulsive disorder. *American Journal of Psychiatry*, 162, 151–161.
- Franklin, M. E., & Foa, E. B. (2011). Treatment of obsessive compulsive disorder. *Annual Review of Clinical Psychology*, 7, 229–243.
- Freeston, M. H., Dugas, M. J., & Ladouceur, R. (1996). Thoughts, images worry, and anxiety. *Cognitive Therapy and Research*, 20, 265–273.
- Frost, R. O., & Steketee, G. (2010). *Stuff: compulsive hoarding and the meaning of things*. New York: Houghton Mifflin Harcourt.
- Frost, R. O., Steketee, G., & Greene, K. A. I. (2003). Cognitive and behavioral treatment of compulsive hoarding. *Brief Treatment and Crisis Intervention*, 3, 323–337.
- Frost, R. O., Steketee, G., & Tolin, D. F. (2012). Diagnosis and assessment of hoarding disorder. *Annual Review of Clinical Psychology*, 8, 219–242.
- Frost, R. O., Tolin, D. F., Steketee, G., Fitch, K., & Selbo-Bruns, A. (2009). Excessive acquisition in hoarding. *Journal of Anxiety Disorders*, 23, 632–639.
- Galea, S., Ahern, J., Resnick, H., Kilpatrick, D., Bucuvalas, M., Gold, J., et al. (2002). Psychological sequelae of the September 11 terrorist attacks in New York City. *New England Journal of Medicine*, 346, 982–987.
- Gentes, E. L., & Ruscio, A. M. (2011). A meta-analysis of the relation of intolerance of uncertainty to symptoms of generalized anxiety disorder, major depressive disorder, and obsessive-compulsive disorder. *Clinical Psychology Review*, 31, 923–933.
- Gerlach, A. L., Wilhelm, F. H., Gruber, K., & Roth, W. T. (2001). Blushing and physiological arousability in social phobia. *Journal of Abnormal Psychology*, 110, 247–258.
- Gilbertson, M. W., Shenton, M. E., Ciszewski, A., Kasai, K., Lasko, N. B., Orr, S. P., et al. (2002). Smaller hippocampal volume predicts pathologic vulnerability to psychological trauma. *Nature Neuroscience*, 5, 1242–1247.
- Gillan, C. M., Morein-Zamir, S., Urcelay, G. P., Sule, A., Voon, V., Apergis-Schoute, A. M., et al. (2014). Enhanced avoidance habits in obsessive-compulsive disorder. *Biological Psychiatry*, 75, 631–638.
- Goldin, P. R., Manber-Ball, T., Werner, K., Heimberg, R., & Gross, J. J. (2009). Neural mechanisms of cognitive reappraisal of negative self-beliefs in social anxiety disorder. *Biological Psychiatry*, 66, 1091–1099.
- Goldstein, A. J., & Chambless, D. L. (1978). A reanalysis of agoraphobic behavior. *Behavior Therapy*, 9, 47–59.
- Goldstein, A. J., de Beurs, E., Chambless, D. L., & Wilson, K. A. (2000). EMDR for panic disorder with agoraphobia: Comparison with waiting list and credible attention-placebo control conditions. *Journal of Consulting and Clinical Psychology*, 68, 947–956.
- Grant, J. E., Odlaug, B. L., & Schreiber, L. R. N. (2014). Pharmacotherapy for obsessive-compulsive and related disorders among adults. In E. A. Storch & D. McKay (Eds.), *Obsessive-Compulsive Disorder and Its Spectrum: A Life-Span Approach*. Washington DC: American Psychological Association.
- Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., et al. (2010). Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey Replication: Associations with first onset of DSM-IV Disorders. *Archives of General Psychiatry*, 67, 113–123.
- Grisham, J. R., Frost, R. O., Steketee, G., Kim, H. J., & Hood, S. (2006). Age of onset of compulsive hoarding. *Journal of Anxiety Disorders*, 20, 675–686.
- Gustad, J., & Phillips, K. A. (2003). Axis I comorbidity in body dysmorphic disorder. *Comprehensive Psychiatry*, 44, 270–276.
- Harvey, A. G., & Bryant, R. A. (2002). Acute stress disorder: A synthesis and critique. *Psychological Bulletin*, 128, 886–902.
- Herman, J. L. (1992). *Trauma and recovery*. New York: Basic Books.
- Hettema, J. M., Neale, M. C., & Kendler, K. S. (2001). A review and meta-analysis of the genetic epidemiology of the anxiety disorders. *American Journal of Psychiatry*, 158, 1568–1578.
- Hettema, J. M., Prescott, C. A., Myers, J. M., Neale, M. C., & Kendler, K. S. (2005). The structure of genetic and environmental risk factors for anxiety disorders in men and women. *Archives of General Psychiatry*, 62, 182–189.
- Hinton, D., Ba, P., Peou, S., & Um, K. (2000). Panic disorder among Cambodian refugees attending a psychiatric clinic. *General Hospital Psychiatry*, 22, 437–444.
- Hinton, E., Um, K., & Ba, P. (2001). Kyol Goeu ('Wind Overload') Part II: Prevalence, characteristics, and mechanisms of Kyol Goeu and near Kyol Goeu episodes of Khmer patients attending a psychiatric clinic. *Transcultural Psychiatry*, 38, 433–460.
- Hirsch, C. R., & Clark, D. M. (2004). Mental imagery and social phobia. In J. Yiend (Ed.), *Cognition, emotion and psychopathology: Theoretical, empirical and clinical directions* (pp. 232–250). Cambridge, UK: Cambridge University Press.
- Hoffman, E. J., & Mathew, S. J. (2008). Anxiety disorders: A comprehensive review of pharmacotherapies. *Mount Sinai Journal of Medicine*, 75, 248–262.

- Hofmann, S. G., & Smits, J. A. (2008). Cognitive-behavioral therapy for adult anxiety disorders: A meta-analysis of randomized placebo-controlled trials. *The Journal of Clinical Psychiatry*, 69, 621–632.
- Hofmann, S. G., Sawyer, A. T., Witt, A. A., & Oh, D. (2010). The effect of mindfulness-based therapy on anxiety and depression: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, 78, 169–183.
- Hollon, S. D., Stewart, M. O., & Strunk, D. (2006). Enduring effects for cognitive behavior therapy in the treatment of depression and anxiety. *Annual Review of Psychology*, 57, 285–315.
- Iervolino, A. C., Perroud, N., Fullana, M. A., Guipponi, M., Cherkas, L., Collier, D. A., et al. (2009). Prevalence and heritability of compulsive hoarding: A twin study. *American Journal of Psychiatry*, 166, 1156–1161.
- IMS Health. (2012). IMS national prescription audit PlusTM. (2011). Downloaded June 2014 from [www.imshealth.com/portal/site/imshealth](http://www.imshealth.com/portal/site/imshealth).
- Indovina, I., Robbins, T. W., Nunez-Elizalde, A. O., Dunn, B. D., & Bishop, S. J. (2011). Fear-conditioning mechanisms associated with trait vulnerability to anxiety in humans. *Neuron*, 69, 563–571.
- Insel, T. R., Scanlan, J., Champoux, M., & Suomi, S. J. (1988). Rearing paradigm in a nonhuman primate affects response to B-CCE challenge. *Psychopharmacology*, 96, 81–86.
- Ipsen, J. C., Sander, C., & Stein, D. J. (2009). Pharmacotherapy and psychotherapy for body dysmorphic disorder. *Cochrane Database of Systematic Reviews* CD005332.
- Jacobsen, L. K., Southwick, S. M., & Kosten, T. R. (2001). Substance use disorders in patients with posttraumatic stress disorder: A review of the literature. *American Journal of Psychiatry*, 158, 1184–1190.
- Johnson, J., Weissman, M. M., & Klerman, G. L. (1992). Service utilization and social morbidity associated with depressive symptoms in the community. *Journal of the American Medical Association*, 267, 1478–1483.
- Kagan, J., & Snidman, N. (1999). Early childhood predictors of adult anxiety disorders. *Biological Psychiatry*, 46, 1536–1541.
- Kapczinski, F., Lima, M. S., Souza, J. S., Cunha, A., & Schmitt, R. (2002). Antidepressants for generalized anxiety disorder. *Cochrane Database of Systematic Reviews*, Issue 2, CD003592.
- Kashdan, T. B., & McKnight, P. E. (2010). The darker side of social anxiety: When aggressive impulsivity prevails over shy inhibition. *Current Directions in Psychological Science*, 19, 47–50.
- Kawakami, N., Shimizu, H., Haratani, T., Iwata, N., & Kitamura, T. (2004). Lifetime and 6-month prevalence of DSM-III-R psychiatric disorders in an urban community in Japan. *Psychiatry Research*, 121, 293–301.
- Keane, T. M., Fairbank, J. A., Caddell, J. M., & Zimering, R. T. (1989). Implosive (flooding) therapy reduces symptoms of PTSD in Vietnam combat veterans. *Behavior Therapy*, 20, 245–260.
- Keane, T. M., Gerardi, R. J., Quinn, S. J., & Litz, B. T. (1992). Behavioral treatment of post-traumatic stress disorder. In S. M. Turner, K. S. Calhoun & H. E. Adams (Eds.), *Handbook of Clinical Behavior Therapy* (2nd ed., pp. 87–97). New York: John Wiley & Sons.
- Keane, T. M., Marshall, A. D., & Taft, C. T. (2006). Posttraumatic stress disorder: Etiology, epidemiology, and treatment outcome. *Annual Review of Clinical Psychology*, 2, 161–197.
- Keane, T. M., Zimering, R. T., & Caddell, J. (1985). A behavioral formulation of posttraumatic stress disorder in Vietnam veterans. *The Behavior Therapist*, 8, 9–12.
- Kenardy, J., & Taylor, C. B. (1999). Expected versus unexpected panic attacks: A naturalistic prospective study. *Journal of Anxiety Disorders*, 13, 435–445.
- Kendler, K. S., Myers, J., & Prescott, C. A. (2002). The etiology of phobias: An evaluation of the stress-diathesis model. *Archives of General Psychiatry*, 59, 242–249.
- Kendler, K. S., Myers, J., Prescott, C. A., & Neale, M. C. (2001). The genetic epidemiology of irrational fears and phobias in men. *Archives of General Psychiatry*, 58, 257–267.
- Kessler, R. C., Crum, R. M., Warner, L. A., Nelson, C. B., et al. (1997). Lifetime co-occurrence of DSM-III-R alcohol dependence with other psychiatric disorders in the National Comorbidity Study. *Archives of General Psychiatry*, 54, 313–321.
- Kidson, M. A., Douglas, J. C., & Holwill, B. J. (1993). Post-traumatic stress disorder in Australian World War II veterans attending a psychiatric outpatient clinic. *The Medical Journal of Australia*, 158(8):563–566.
- Kim, H. J., Steketee, G., & Frost, R. O. (2001). Hoarding by elderly people. *Health and Social Work*, 26, 176–184.
- Kim, M. J., Loucks, R. A., Palmer, A. L., Brown, A. C., Solomon, K. M., Marchante, A. N., et al. (2011). The structural and functional connectivity of the amygdala: From normal emotion to pathological anxiety. *Behavioural Brain Research*, 223, 403–410.
- Kirmayer, L. J. (2001). Cultural variations in the clinical presentation of depression and anxiety: Implications for diagnosis and treatment. *Journal of Clinical Psychiatry*, 62 (Suppl. 13), 22–28.
- Koenen, K. C., Moffitt, T. E., Poulton, R., Martin, J., & Caspi, A. (2007). Early childhood factors associated with the development of post-traumatic stress disorder: Results from a longitudinal birth cohort. *Psychological Medicine*, 37, 181–192.
- Kremen, W. S., Koenen, K. C., Boake, C., Purcell, S., Eisen, S. A., Franz, C. E., et al. (2007). Pretrauma cognitive ability and risk for posttraumatic stress disorder. *Archives of General Psychiatry*, 64, 361–368.
- Ladoceur, R., Dugas, M. J., Freeston, M. H., Leger, E., Gagnon, F., & Thibodeau, N. (2000). Efficacy of a new cognitive-behavioral treatment for generalized anxiety disorder: Evaluation in a controlled clinical trial. *Journal of Consulting and Clinical Psychology*, 68, 957–996.
- Lambrou, C., Veale, D., & Wilson, G. (2011). The role of aesthetic sensitivity in body dysmorphic disorder. *Journal of Abnormal Psychology*, 120, 443–453.

- Laugesen, N., Dugas, M. J., & Bukowski, W. M. (2003). Understanding adolescent worry: The application of a cognitive model. *Journal of Abnormal Child Psychology*, 31, 55–64.
- LeBeau, R. T., Glenn, D., Liao, B., Wittchen, H. U., Beesdo-Baum, K., Ollendick, T., et al. (2010). Specific phobia: A review of DSM-IV specific phobia and preliminary recommendations for DSM-V. *Depression and Anxiety*, 27, 148–167.
- Leon, A. C., Portera, L., & Weissman, M. M. (1995). The social costs of anxiety disorders. *British Journal of Psychiatry*, 166 (Suppl. 27), 19–22.
- Liebowitz, M. R., Heimberg, R. G., Fresco, D. M., Travers, J., & Stein, M. B. (2000). Social phobia or social anxiety disorder: What's in a name? *Archives of General Psychiatry*, 57, 191–192.
- Lindemann, E., & Finesinger, I. E. (1938). The effect of adrenalin and mecholyl in states of anxiety in psychoneurotic patients. *American Journal of Psychiatry*, 95, 353–370.
- Lipsitz, J. D., Mannuzza, S., Klein, D. F., Ross, D. C., & Fyer, A. J. (1999). Specific phobia 10–16 years after treatment. *Depression and Anxiety*, 10, 105–111.
- Lissek, S., Powers, A. S., McClure, E. B., Phelps, E. A., Woldehawariat, G., Grillon, C., et al. (2005). Classical fear conditioning in the anxiety disorders: A meta-analysis. *Behaviour Research and Therapy*, 43, 1391–1424.
- Litz, B. T., Gray, M. J., Bryant, R. A., & Adler, A. B. (2002). Early intervention for trauma: Current status and future directions. *Clinical Psychology: Science and Practice*, 9, 112–134.
- Lohr, J., Tolin, D. F., & Lilienfeld, S. O. (1998). Efficacy of eye movement desensitization and reprocessing: Implications for behavior therapy. *Behavior Therapy*, 29, 123–156.
- MacLeod, C., & Mathews, A. (2012). Cognitive bias modification approaches to anxiety. *Annual Review of Clinical Psychology*, 8, 189–217.
- Malizia, A. L. (2003). Brain imaging and anxiety disorders. In D. Nutt & J. Ballenger (Eds.), *Anxiety Disorders* (pp. 201–228). Malden, MA: Blackwell.
- Mancebo, M. C., Eisen, J. L., Sibrava, N. J., Dyck, I. R., & Rasmussen, S. A. (2011). Patient utilization of cognitive-behavioral therapy for OCD. *Behavior Therapy*, 42, 399–412.
- Maren, S., Phan, K. L., & Liberzon, I. (2013). The contextual brain: Implications for fear conditioning, extinction and psychopathology. *Nature Reviews Neuroscience*, 14, 417–428.
- Margraf, J., Ehlers, A., & Roth, W. T. (1986). Sodium lactate infusions and panic attacks: A review and critique. *Psychosomatic Medicine*, 48, 23–51.
- Martin, J. H. (1996). *Neuroanatomy: Text and atlas* (4th ed.). New York: McGraw-Hill Medical.
- Mataix-Cols, D., Marks, I. M., Greist, J. H., Kobak, K. A., & Baer, L. (2002). Obsessive-compulsive symptom dimensions as predictors of compliance with and response to behaviour therapy: Results from a controlled trial. *Psychotherapy and Psychosomatics*, 71, 255–262.
- McGuire, P. K., Bench, C. J., Frith, C. D., & Marks, I. M. (1994). Functional anatomy of obsessive-compulsive phenomena. *British Journal of Psychiatry*, 164, 459–468.
- McNally, R. J. (1987). Preparedness and phobias: A review. *Psychological Bulletin*, 101, 283–303.
- McNally, R. J. (1997). Atypical phobias. In G. C. L. Davey (Ed.), *Phobias: A handbook of theory, research and treatment* (pp. 183–199). Chichester, UK: John Wiley & Sons.
- McNally, R. J. (2003). *Remembering trauma*. Cambridge, MA: Belknap Press of Harvard University Press.
- McNally, R. J., Caspi, S. P., Riemann, B. C., & Zeitlin, S. B. (1990). Selective processing of threat cues in posttraumatic stress disorder. *Journal of Abnormal Psychology*, 99, 398–402.
- McTeague, L. M., & Lang, P. J. (2012). The anxiety spectrum and the reflex physiology of defense: From circumscribed fear to broad distress. *Depression and Anxiety*, 29, 264–281.
- Mennin, D. S., Heimberg, R. G., & Turk, C. L. (2004). Clinical presentation and diagnostic features. In R. G. Heimberg, C. L. Turk & D. S. Mennin (Eds.), *Generalized anxiety disorder* (pp. 3–28). New York: Guilford Press.
- Mennin, D. S., Heimberg, R. G., Turk, C. L., & Fresco, D. M. (2002). Applying an emotion regulation framework to integrative approaches to generalized anxiety disorder. *Clinical Psychology: Science and Practice*, 9, 135–141.
- Menzies, L., Chamberlain, S. R., Laird, A. R., Thelen, S. M., Sahakian, B. J., & Bullmore, E. T. (2008). Integrating evidence from neuroimaging and neuropsychological studies of obsessive-compulsive disorder: The orbitofronto-striatal model revisited. *Neuroscience and Biobehavioral Reviews*, 32, 525–549.
- Meyer, V. (1966). Modification of expectations in cases with obsessional rituals. *Behaviour Research and Therapy*, 4, 273–280.
- Michael, T., Blechert, J., Vriends, N., Margraf, J., & Wilhelm, F. H. (2007). Fear conditioning in panic disorder: Enhanced resistance to extinction. *Journal of Abnormal Psychology*, 116, 612–617.
- Mineka, S., & Öhman, A. (2002). Born to fear: Nonassociative vs. associative factors in the etiology of phobias. *Behaviour Research and Therapy*, 40, 173–184.
- Mineka, S., & Sutton, J. (2006). Contemporary learning theory perspectives on the etiology of fear and phobias. In M. G. Craske, D. Hermans & D. Vansteenwegen (Eds.), *Fear and learning: From basic processes to Clinical implications* (pp. 75–97). Washington DC: American Psychological Association.
- Mineka, S., & Zinbarg, R. (1998). Experimental approaches to the anxiety and mood disorders. In J. G. Adair, D. Belanger & K. L. Dion (Eds.), *Advances in psychological science, Volume 1: Social personal and cultural aspects* (pp. 429–454). Hove, UK: Psychology Press.

- Mineka, S., & Zinbarg, R. (2006). A contemporary learning theory perspective on the etiology of anxiety disorders: It's not what you thought it was. *The American Psychologist*, 61, 10–26.
- Mitchell, J. T., & Everly, G. S., Jr. (2000). Critical incident stress management and critical incident stress debriefings: Evolutions, effects and outcomes. In J. P. Wilson & B. Raphael (Eds.), *Psychological debriefing: Theory, practice and evidence* (pp. 71–90). New York: Cambridge University Press.
- Mitte, K. (2005). Meta-analysis of cognitive-behavioral treatments for generalized anxiety disorder: A comparison with pharmacotherapy. *Psychological Bulletin*, 131, 785–795.
- Monk, C. S., Nelson, E. E., McClure, E. B., Mogg, K., Bradley, B. P., Leibenluft, E., et al. (2006). Ventrolateral prefrontal cortex activation and attentional bias in response to angry faces in adolescents with generalized anxiety disorder. *American Journal of Psychiatry*, 163, 1091–1097.
- Monzani, B., Rijdsdijk, F., Iervolino, A. C., Anson, M., Cherkas, L., & Mataix-Cols, D. (2012). Evidence for a genetic overlap between body dysmorphic concerns and obsessive-compulsive symptoms in an adult female community twin sample. *American Journal of Medical Genetics Part B, Neuropsychiatric genetics*, 159B, 376–382.
- Moulding, R., & Kyrios, M. (2006). Anxiety disorders and control related beliefs: The exemplar of obsessive-compulsive disorder (OCD). *Clinical Psychology Review*, 26(5), 573–583. <http://dx.doi.org/10.1016/j.cpr.2006.01.009>
- Mower, O. H. (1947). On the dual nature of learning: A reinterpretation of “conditioning” and “problem-solving”. *Harvard Educational Review*, 17, 102–148.
- Moylan, S., Staples, J., Ward, S. A., Rogerson, J., Stein, D. J., & Berk, M. (2011). The efficacy and safety of alprazolam versus other benzodiazepines in the treatment of panic disorder. *Journal of Clinical Psychopharmacology*, 31, 647–652.
- Muroff, J., Levis, M. E., & Bratotis, C. (2014). Hoarding disorder. In E. A. Storch & D. McKay (Eds.), *Obsessive-compulsive disorder and its spectrum: A life-span approach* (pp. 117–140). Washington, DC: American Psychological Association.
- Nay, W., Brown, R., & Roberson-Nay, R. (2013). Longitudinal course of panic disorder with and without agoraphobia using the National Epidemiologic Survey on Alcohol and Related Conditions (NESARC). *Psychiatry Research*, 208, 54–61.
- Neumeister, A., Daher, R. J., & Charney, D. S. (2005). Anxiety disorders: noradrenergic neurotransmission. *Handbook of Experimental Pharmacology*, 169, 205–223.
- Newman, D. L., Moffitt, T. E., Caspi, A., & Silva, P. A. (1998). Comorbid mental disorders: Implications for treatment and sample selection. *Journal of Abnormal Psychology*, 107, 305–311.
- Newman, J. P., Schmitt, W. A., & Voss, W. D. (1997). The impact of motivationally neutral cues on psychopathic individuals: Assessing the generality of the response modulation hypothesis. *Journal of Abnormal Psychology*, 196, 563–575.
- Norton, P. J., & Price, E. C. (2007). A meta-analytic review of adult cognitive-behavioral treatment outcome across the anxiety disorders. *Journal of Nervous and Mental Disease*, 195, 521–531.
- Öhman, A., & Mineka, A. (2003). The malicious serpent: Snakes as a prototypical stimulus for an evolved module of fear. *Current Directions in Psychological Science*, 12, 5–9.
- Öhman, A., & Soares, J. J. F. (1994). “Unconscious anxiety”: Phobic responses to masked stimuli. *Journal of Abnormal Psychology*, 103, 231–240.
- Öhman, A., Flykt, A., & Esteves, F. (2001). Emotion drives attention: Detecting the snake in the grass. *Journal of Experimental Psychology: General*, 137, 466–478.
- Olatunji, B. O., Cisler, J. M., & Tolin, D. F. (2007). Quality of life in the anxiety disorders: A meta-analytic review. *Clinical Psychology Review*, 27, 572–581.
- Olatunji, B. O., Etzel, E. N., Tomarken, A. J., Ciesielski, B. G., & Deacon, B. (2011). The effects of safety behaviors on health anxiety: An experimental investigation. *Behaviour Research and Therapy*, 49, 719–728.
- Olatunji, B. O., Moretz, M. W., & Zlomke, K. R. (2010). Linking cognitive avoidance and GAD symptoms: The mediating role of fear of emotion. *Behaviour Research and Therapy*, 48, 435–441.
- Olf, M., Langeland, W. L., Draijer, N., & Gersons, B. P. R. (2007). Gender differences in posttraumatic stress disorder. *Psychological Bulletin*, 133, 183–204.
- Orr, S. P., Metzger, L. J., Lasko, N. B., Macklin, M. L., Hu, F. B., Shalev, A. Y., et al. (2003). Physiologic responses to sudden, loud tones in monozygotic twins discordant for combat exposure: Association with posttraumatic stress disorder. *Archives of General Psychiatry*, 60, 283–288.
- Ozer, E. J., Best, S. R., Lipsey, T. L., & Weiss, D. S. (2003). Predictors of posttraumatic stress disorder and symptoms in adults: A meta-analysis. *Psychological Bulletin*, 129, 52–73.
- Patronek, G. J., & Nathanson, J. N. (2009). A theoretical perspective to inform assessment and treatment strategies for animal hoarders. *Clinical Psychology Review*, 29, 274–281.
- Perkonig, A., Pfister, H., Stein, M. B., Hofler, M., Lieb, R., Maercker, A., et al. (2005). Longitudinal course of posttraumatic stress disorder and posttraumatic stress. *American Journal of Psychiatry*, 162, 1320–1327.
- Perugi, G., Akiskal, H. S., Giannotti, D., Frare, F., Di Vaio, S., & Cassano, G. B. (1997). Gender-related differences in body dysmorphic disorder. *Journal of Nervous and Mental Disease*, 185, 578–582.
- Phillips, K. A. (2005). *The broken mirror: Understanding and treating body dysmorphic disorder*. New York: Oxford University Press.
- Phillips, K. A. (2006). “I look like a monster”: Pharmacotherapy and cognitive-behavioral therapy for body dysmorphic disorder. In R. L. Spitzer, M. B. First, J. B. W. Williams & M. Gibbon (Eds.), *DSM-IV-TR Case Book* (Vol. 2) *Experts tell how they treated their own patients* (pp. 263–276). Washington, DC: American Psychiatric Publishing.

- Phillips, K. A., Menard, W., Quinn, E., Didie, E. R., & Stout, R. L. (2013). A 4-year prospective observational follow-up study of course and predictors of course in body dysmorphic disorder. *Psychological Medicine*, 43, 1109–1117.
- Phillips, K. A., Pinto, A., Hart, A. S., Coles, M. E., Eisen, J. L., Menard, W., et al. (2012). A comparison of insight in body dysmorphic disorder and obsessive-compulsive disorder. *Journal of Psychiatric Research*, 46, 1293–1299.
- Phillips, K. A., Stein, D. J., Rauch, S. L., Hollander, E., Fallon, B. A., Barsky, A., et al. (2010). Should an obsessive-compulsive spectrum grouping of disorders be included in DSM-V? *Depression and Anxiety*, 27, 528–555.
- Phillips, K. A., Wilhelm, S., Koran, L. M., Didie, E. R., Fallon, B. A., Feusner, J., et al. (2010). Body dysmorphic disorder: Some key issues for DSM-V. *Depression and Anxiety*, 27, 573–591.
- Phoenix Australia. (n.d.). *Home page*. Retrieved from <http://phoenixaustralia.org>
- Pineles, S. L., & Mineka, S. (2005). Attentional biases to internal and external sources of potential threat in social anxiety. *Journal of Abnormal Psychology*, 114, 314–318.
- Pole, N., Neylan, T. C., Otte, C., Henn-Hasse, C., Metzler, T. J., & Marmar, C. R. (2009). Prospective prediction of posttraumatic stress disorder symptoms using fear potentiated auditory startle responses. *Biological Psychiatry*, 65, 235–240.
- Powers, M. B., Halpern, J. M., Ferenschak, M. P., Gillihan, S. J., & Foa, E. B. (2010). A meta-analytic review of prolonged exposure for posttraumatic stress disorder. *Clinical Psychology Review*, 30, 635–641.
- Rääbus, C. (2016, March 21). Tasmanian boxing champ Luke Jackson: My daily fight with obsessive compulsive disorder. *ABC News*. Retrieved from [www.abc.net.au/news/2016-03-21/boxing-champion-luke-jackson-battling-ocd/7263030](http://www.abc.net.au/news/2016-03-21/boxing-champion-luke-jackson-battling-ocd/7263030)
- Rachman, S. J. (1977). The conditioning theory of fear acquisition: A critical examination. *Behaviour Research and Therapy*, 15, 375–387.
- Rachman, S. J., & DeSilva, P. (1978). Abnormal and normal obsessions. *Behaviour Research and Therapy*, 16, 233–248.
- Rademaker, A. R., van Zuiden, M., Vermetten, E., & Geuze, E. (2011). Type D personality and the development of PTSD symptoms: A prospective study. *Journal of Abnormal Psychology*, 120, 299–307.
- Rapoport, J. L., Swedo, S. E., & Leonard, H. L. (1992). Childhood obsessive compulsive disorder. *Journal of Clinical Psychiatry*, 53, 11–16.
- Rapoport, M. J., Lanctot, K. L., Streiner, D. L., Bedard, M., Vingilis, E., Murray, B., et al. (2009). Benzodiazepine use and driving: A meta-analysis. *Journal of Clinical Psychiatry*, 70, 663–673.
- Rauch, S. L., Whalen, P. J., Shin, L. M., McInerney, S. C., Macklin, M. L., Lasko, N. B., et al. (2000). Exaggerated amygdala response to masked facial stimuli in posttraumatic stress disorder: A functional MRI study. *Biological Psychiatry*, 47, 769–776.
- Rebecca Gibney opens up about her panic attacks and battle with depression. (2014, April 21). *Herald Sun*. Retrieved from [www.heraldsun.com.au/entertainment/celebrity/rebecca-gibney-opens-up-about-her-panic-attacks-and-battle-with-depression/news-story/18e5b155ac028744f8360fc29dbde82f](http://www.heraldsun.com.au/entertainment/celebrity/rebecca-gibney-opens-up-about-her-panic-attacks-and-battle-with-depression/news-story/18e5b155ac028744f8360fc29dbde82f)
- Reichborn-Kjennerud, T., Czajkowski, N., Neale, M. C., Ørstavik, R. E., Torgersen, S., Tambs, K., et al. (2007). Genetic and environmental influences on dimensional representations of DSM-IV cluster C personality disorders: A population-based multivariate twin study. *Psychological Medicine*, 37, 645–653.
- Resick, P. A., Bovin, M. J., Calloway, A. L., Dick, A. M., King, M. W., Mitchell, K. S., et al. (2012). A critical evaluation of the complex PTSD literature: Implications for DSM-5. *Journal of Traumatic Stress*, 25, 241–251.
- Resick, P. A., Nishith, P., & Griffin, M. G. (2003). How well does cognitive-behavioral therapy treat symptoms of complex PTSD? An examination of child sexual abuse survivors within a clinical trial. *CNS Spectrums*, 8, 351–355.
- Resick, P. A., Nishith, P., Weaver, T. L., Astin, M. C., & Feuer, C. A. (2002). A comparison of cognitive-processing therapy with prolonged exposure and a waiting condition for the treatment of chronic posttraumatic stress disorder in female rape victims. *Journal of Consulting and Clinical Psychology*, 70, 867–879.
- Resick, P. A., Suvak, M. K., Johnides, B. D., Mitchell, K. S., & Iverson, K. M. (2012). The impact of dissociation on PTSD treatment with cognitive processing therapy. *Depression and Anxiety*, 29, 718–730.
- Roemer, L., Lee, J. K., Salters-Pedneault, K., Erisman, S. M., Orsillo, S. M., & Mennin, D. S. (2009). Mindfulness and emotion regulation difficulties in generalized anxiety disorder: Preliminary evidence for independent and overlapping contributions. *Behavior Therapy*, 40, 142–154.
- Roemer, L., Molina, S., & Borkovec, T. D. (1997). An investigation of worry content among generally anxious individuals. *Journal of Nervous and Mental Disease*, 185, 314–319.
- Roest, A. M., Martens, E. J., de Jonge, P., & Denollet, J. (2010). Anxiety and risk of incident coronary heart disease: A meta-analysis. *Journal of the American College of Cardiology*, 56, 38–46.
- Rosa-Alcazar, A. I., Sanchez-Meca, J., Gomez-Conesa, A., & Marin-Martinez, F. (2008). Psychological treatment of obsessive-compulsive disorder: A meta-analysis. *Clinical Psychology Review*, 28, 1310–1325.
- Rosen, G. M., & Davison, G. C. (2003). Psychology should list empirically supported principles of change (ESPs) and not credential trademarked therapies or other treatment packages. *Behavior Modification*, 27, 300–312.
- Rotge, J. Y., Guehl, D., Dilharreguy, B., Tignol, J., Bioulac, B., Allard, M., et al. (2009). Meta-analysis of brain volume changes in obsessive-compulsive disorder. *Biological Psychiatry*, 65, 75–83.
- Rothbaum, B. O., Anderson, P., Zimand, E., Hodges, L., Lang, D., & Wilson, J. (2006). Virtual reality exposure therapy and standard (in vivo) exposure therapy in the treatment of fear of flying. *Behavior Therapy*, 37, 80–90.
- Rothbaum, B. O., Foa, E. B., Murdock, T., Riggs, D. S., & Walsh, W. (1992). A prospective examination of posttraumatic stress disorder in rape victims. *Journal of Traumatic Stress*, 5, 455–475.

- Rubin, D. C., Berntsen, D., & Bohni, M. K. (2008). A memory-based model of posttraumatic stress disorder: Evaluating basic assumptions underlying the PTSD diagnosis. *Psychological Review*, 115, 985–1011.
- Ruscio, A. M., Brown, T. A., Chiu, W. T., Sareen, J., Stein, M. B., & Kessler, R. C. (2008). Social fears and social phobia in the USA: Results from the National Comorbidity Survey Replication. *Psychological Medicine*, 38, 15–28.
- Salkovskis, P. M. (1996). Cognitive-behavioral approaches to understanding obsessional problems. In R. M. Rapee (Ed.), *Current controversies in anxiety disorders*. New York: Guilford Press.
- Samuels, J. F. (2009). Recent advances in the genetics of obsessive-compulsive disorder. *Current Psychiatry Reports*, 11, 277–282.
- Samuels, J. F., Bienvenu, O. J., 3rd, Pinto, A., Fyer, A. J., McCracken, J. T., Rauch, S. L., et al. (2007). Hoarding in obsessive-compulsive disorder: Results from the OCD Collaborative Genetics Study. *Behaviour Research and Therapy*, 45, 673–686.
- Sareen, J., Cox, B. J., Afifi, T. O., de Graaf, R., Asmundson, G. J. G., ten Have, M., et al. (2005). Anxiety disorders and risk for suicidal ideation and suicide attempts: A population-based longitudinal study of adults. *Archives of General Psychiatry*, 62, 1249–1257.
- Saxena, S., Brody, A. L., Maidment, K. M., & Baxter, L. R. J. (2007). Paroxetine treatment of compulsive hoarding. *Journal of Psychiatric Research*, 41, 481–487.
- Schmidt, N. B., Richey, J. A., Buckner, J. D., & Timpano, K. R. (2009). Attention training for generalized social anxiety disorder. *Journal of Abnormal Psychology*, 118, 5–14.
- Schweitzer, E., Rickels, K., Case, G., & Greenblatt, D. J. (1990). Long-term therapeutic use of benzodiazapines: Effects of gradual taper. *Archives of General Psychiatry*, 47, 908–915.
- Seedat, S., & Matsunaga, H. (2007). Cross-national and ethnic issues in OC spectrum disorders. *CNS Spectrums*, 12, 392–400.
- Seidler, G. H., & Wagner, F. E. (2006). Comparing the efficacy of EMDR and trauma-focused cognitive-behavioral therapy in the treatment of PTSD: A meta-analytic study. *Psychological Medicine*, 36, 1515–1522.
- Seligman, M. E. P. (1971). Phobias and preparedness. *Behavior Therapy*, 2, 307–320.
- Shapiro, F. (1999). Eye movement desensitization and reprocessing (EMDR) and the anxiety disorders: Clinical and research implications of an integrated psychotherapy treatment. *Journal of Anxiety Disorders*, 13, 35–67.
- Shin, L. M., & Liberzon, I. (2010). The neurocircuitry of fear, stress, and anxiety disorders. *Neuropsychopharmacology*, 35, 169–191.
- Simon, G., Ormel, J., VonKroff, M., & Barlow, W. (1995). Health care costs associated with depressive and anxiety disorders in primary care. *American Journal of Psychiatry*, 152, 352–357.
- Smith, D. (2012). *Monkey mind*. New York: Simon & Schuster.
- Staugaard, S. R. (2010). Threatening faces and social anxiety: A literature review. *Clinical Psychology Review*, 30, 669–690.
- Stein, D. J., Aguilar-Gaxiola, S., Alonso, J., Bruffaerts, R., De Jonge, P., Liu, Z., et al. (2014). Associations between mental disorders and subsequent onset of hypertension. *General Hospital Psychiatry*, 36, 142–149.
- Stein, D. J., Ipser, J. C., & Balkom, A. J. (2004). Pharmacotherapy for social anxiety disorder. *Cochrane Database of Systematic Reviews*, Issue 4, CD001206.
- Stein, D. J., Ipser, J. C., & Seedat, S. (2000). Pharmacotherapy for post traumatic stress disorder (PTSD). *Cochrane Database of Systematic Reviews*, Issue 4, CD002795.
- Stein, D. J., Koenen, K. C., Friedman, M. J., Hill, E., McLaughlin, K. A., Petukhova, M., et al. (2013). Dissociation in posttraumatic stress disorder: Evidence from the World Mental Health surveys. *Biological Psychiatry*, 73, 302–312.
- Steketee, G., & Barlow, D. H. (Eds.). (2004). *Obsessive-compulsive disorder*. New York: Guilford Press.
- Steketee, G., & Frost, R. O. (2003). Compulsive hoarding: Current status of the research. *Clinical Psychology Review*, 23, 905–927.
- Steketee, G., Frost, R. O., Tolin, D. F., Rasmussen, J., & Brown, T. A. (2010). Waitlist-controlled trial of cognitive behavior therapy for hoarding disorder. *Depression and Anxiety*, 27, 476–484.
- Stern, R. S., & Cobb, J. P. (1978). Phenomenology of obsessive-compulsive neurosis. *British Journal of Psychiatry*, 132, 233–234.
- Stopa, L., & Clark, D. M. (2000). Social phobia and interpretation of social events. *Behaviour Research and Therapy*, 38, 273–283.
- Stossel, S. (2014, January/February). Surviving anxiety. *Atlantic Monthly*, 1–6.
- Sullivan, G. M., Coplan, J. D., Kent, J. M., & Gorman, J. M. (1999). The noradrenergic system in pathological anxiety: A focus on panic with relevance to generalized anxiety and phobias. *Biological Psychiatry*, 46, 1205–1218.
- Suzuki, K., Takei, N., Kawai, M., Minabe, Y., & Mori, N. (2003). Is taijin kyofusho a culture-bound syndrome? *American Journal of Psychiatry*, 160(7), 1358.
- Swain, J., Koszycki, D., Shlik, J., & Bradwein, J. (2003). Pharmacological challenge agents in anxiety. In D. Nutt & J. C. Ballenger (Eds.), *Anxiety Disorders* (pp. 269–295). Malden, MA: Blackwell.
- Tambs, K., Czajkowski, N., Roysamb, E., Neale, M. C., Reichborn-Kjennerud, T., Aggen, S. H., et al. (2009). Structure of genetic and environmental risk factors for dimensional representations of DSM-IV anxiety disorders. *British Journal of Psychiatry*, 195, 301–307.
- Taylor, C. B., Hayward, C., King, R., Ehlers, A., Margraf, J., Maddock, R., et al. (1990). Cardiovascular and symptomatic reduction effects of alprazolam and imipramine in patients with panic disorder: Results of a double-blind, placebo-controlled trial. *Journal of Clinical Psychopharmacology*, 10, 112–118.
- Taylor, S., Jang, K. L., & Asmundson, G. J. (2010). Etiology of obsessions and compulsions: A behavioral-genetic analysis. *Journal of Abnormal Psychology*, 119, 672–682.
- Tedeschi, R. G., Park, C. L., & Calhoun, L. G. (1998). Posttraumatic growth: Conceptual issues. In R. G. Tedeschi, C. L. Park & L. G. Calhoun (Eds.), *Posttraumatic growth: Positive changes in the aftermath of trauma* (pp. 1–22). Thousand Oaks, CA: Sage Press.

- Timpano, K. R., Broman-Fulks, J. J., Glaesmer, H., Exner, C., Rief, W., Olatunji, B. O., et al. (2013). A taxometric exploration of the latent structure of hoarding. *Psychological Assessment*, 25, 194–203.
- Tolin, D. F., & Foa, E. B. (2006). Sex differences in trauma and posttraumatic stress disorder: A quantitative review of 25 years of research. *Psychological Bulletin*, 132, 959–992.
- Tolin, D. F., & Villavicencio, A. (2011). Inattention, but not OCD, predicts the core features of hoarding disorder. *Behaviour Research and Therapy*, 49, 120–125.
- Tolin, D. F., Frost, R. O., Steketee, G., Gray, K., & Fitch, K. (2008). The economic and social burden of compulsive hoarding. *Psychiatry Research*, 160, 200–211.
- Tolin, D. F., Kiehl, K. A., Worhunsky, P., Book, G. A., & Maltby, N. (2009). An exploratory study of the neural mechanisms of decision making in compulsive hoarding. *Psychological Medicine*, 39, 325–336.
- Tolin, D. F., Stevens, M. C., Villavicencio, A. L., Norberg, M. M., Calhoun, V. D., Frost, R. O., et al. (2012). Neural mechanisms of decision making in hoarding disorder. *Archives of General Psychiatry*, 69, 832–841.
- Tompkins, M. A., & Hartl, T. L. (2009). *Digging out: Helping your loved one manage clutter, hoarding, and compulsive acquiring*. Oakland, CA: New Harbinger.
- Trinder, H., & Salkovskis, P. M. (1994). Personally relevant intrusions outside the laboratory: Long-term suppression increases intrusion. *Behaviour Research and Therapy*, 32, 833–842.
- True, W. R., Rice, J., Eisen, S. A., Heath, A. C., Godlberg, J., Lyons, M. J., et al. (1993). A twin study of genetic and environmental contributions to liability for posttraumatic stress symptoms. *Archives of General Psychiatry*, 50, 257–264.
- Turner, S. M., Beidel, D. C., & Townsley, R. M. (1990). Social phobia: Relationship to shyness. *Behaviour Research and Therapy*, 28, 297–305.
- Van Oppen, P., de Haan, E., Van Balkom, A. J. L. M., Spinhoven, P., Hoogdiun, K., & van Dyck, R. (1995). Cognitive therapy and exposure in vivo in the treatment of obsessive compulsive disorder. *Behaviour Research and Therapy*, 33, 379–390.
- Veale, D. (2000). Outcome of cosmetic surgery and “DIY” surgery in patients with body dysmorphic disorder. *Psychiatric Bulletin*, 24, 218–221.
- Veale, D. (2004). Advances in a cognitive behavioural model of body dysmorphic disorder. *Body Image*, 1, 113–125.
- Vogt, D., Smith, B., Elwy, R., Martin, J., Schultz, M., Drainoni, M. L., et al. (2011). Predeployment, deployment, and postdeployment risk factors for posttraumatic stress symptomatology in female and male OEF/OIF veterans. *Journal of Abnormal Psychology*, 120, 819–831.
- Vøllestad, J., Nielsen, M. B., & Nielsen, G. H. (2012). Mindfulness- and acceptance-based interventions for anxiety disorders: A systematic review and meta-analysis. *British Journal of Clinical Psychology*, 51, 239–260.
- Voon, V., Derbyshire, K., Ruck, C., Irvine, M. A., Worbe, Y., Enander, J., et al. (2014, in press). Disorders of compulsivity: A common bias towards learning habits. *Molecular Psychiatry*.
- Wegner, D. M., Schneider, D. J., Carter, S. R., & White, T. L. (1987). Paradoxical effects of thought suppression. *Journal of Personality and Social Psychology*, 53, 5–13.
- Weierich, M. R., & Nock, M. K. (2008). Posttraumatic stress symptoms mediate the relation between childhood sexual abuse and nonsuicidal self-injury. *Journal of Consulting and Clinical Psychology*, 76, 39–44.
- Wells, A. (1998). Cognitive therapy of social phobia. In N. Tarrier, A. Wells & G. Haddock (Eds.), *Treating complex cases: The cognitive-behavioural approach* (pp. 1–26). Chichester, UK: John Wiley & Sons.
- Whisman, M. A., & Bruce, M. L. (1999). Marital dissatisfaction and incidence of major depressive episode in a community sample. *Journal of Abnormal Psychology*, 108, 674–678.
- Whisman, M. A., Sheldon, C. T., & Goering, P. (2000). Psychiatric disorders and dissatisfaction with social relationships: does type of relationship matter? *Journal of Abnormal Psychology*, 109, 803–808.
- Wilhelm, S., Buhlmann, U., Hayward, L. C., Greenberg, J. L., & Dimaite, R. (2010). A cognitive-behavioral treatment approach for body dysmorphic disorder. *Cognitive and Behavioral Practice*, 17, 241–247.
- Wilhelm, S., Phillips, K. A., Didie, E., Buhlmann, U., Greenberg, J. L., Fama, J. M., et al. (2014). Modular cognitive-behavioral therapy for body dysmorphic disorder: A randomized controlled trial. *Behavior Therapy*, 45, 314–327.
- Williams, J. M. G., Watts, F. N., MacLeod, C., & Mathews, A. (1997). *Cognitive psychology and emotional disorders* (2nd ed.). New York: John Wiley & Sons.
- Williams, J., Hadjistavropoulos, T., & Sharpe, D. (2006). A meta-analysis of psychological and pharmacological treatments for body dysmorphic disorder. *Behaviour Research and Therapy*, 44, 99–111.
- Wincze, J. P., Steketee, G., & Frost, R. O. (2007). Categorization in compulsive hoarding. *Behaviour Research and Therapy*, 45, 63–72.
- Wittchen, H. U., Gloster, A. T., Beesdo-Baum, K., Fava, G. A., & Craske, M. G. (2010). Agoraphobia: A review of the diagnostic classificatory position and criteria. *Depression and Anxiety*, 27, 113–133.
- Wolf, E. J., Miller, M. W., Reardon, A. F., Ryabchenko, K. A., Castillo, D., & Freund, R. (2012). A latent class analysis of dissociation and posttraumatic stress disorder: Evidence for a dissociative subtype. *Archives of General Psychiatry*, 69, 698–705.
- Woody, E. Z., & Szechtman, H. (2011). Adaptation to potential threat: The evolution, neurobiology, and psychopathology of the security motivation system. *Neuroscience and Biobehavioral Reviews*, 35, 1019–1033.

- Woody, S. R., & Teachman, B. A. (2000). Intersection of disgust and fear: Normative and pathological views. *Clinical Psychology: Science and Practice*, 7, 291–311.
- Yehuda, R., & LeDoux, J. (2007). Response variation following trauma: A translational neuroscience approach to understanding PTSD. *Neuron*, 56, 19–32.
- Yerkes, R. M., & Dodson, J. D. (1908). The relation of strength of stimulus to rapidity of habit formation. *Journal of Comparative and Neurological Psychology*, 18, 459–482.
- Yonkers, K. A., Bruce, S. E., Dyck, I. R., & Keller, M. B. (2003). Chronicity, relapse, and illness-course of panic disorder, social phobia, and generalized anxiety disorder: Findings in men and women from 8 years of follow-up. *Depression and Anxiety*, 17, 173–179.
- Zane, M. D. (1984). Psychoanalysis and contextual analysis of phobias. *Journal of the American Academy of Psychoanalysis*, 12, 553–568.
- Zanov, M. V., & Davison, G. C. (2009). A conceptual and empirical review of 25 years of cognitive assessment using the Articulated Thoughts in Simulated Situations (ATSS) think-aloud paradigm. *Cognitive Therapy and Research*, 34, 282–291.
- Zanov, M. V., & Davison, G. C. (2010). A conceptual and empirical review of 25 years of cognitive assessment using the Articulated Thoughts in Simulated Situations (ATSS) think-aloud paradigm. *Cognitive Therapy and Research*, 34, 282–291.
- Zohar, A. H., & Felz, L. (2001). Ritualistic behavior in young children. *Journal of Abnormal Child Psychology*, 29, 121–128.

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## CHAPTER 5

# Dissociative disorders, and somatic symptom and related disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 5.1** summarise the symptoms, aetiological models and available treatments for dissociative disorders
  - 5.2** summarise the symptoms, aetiology and available treatments for somatic symptom and related disorders.
-

## OPENING SCENARIO

Hanna Q, a 21-year-old single Australian woman from an educated middle-class family, was referred to Dr Middleton by a psychiatrist colleague who had been seeing her regularly for a year. The referral letter stated, 'She has had several hospitalisations with a diagnosis of depression, [and] borderline personality disorder with intense suicidal thoughts. I feel her diagnosis is most consistent with dissociation, derealisation and depersonalisation problems. During the time that I have been seeing her, nothing has happened at all in therapy'. It was added that she attended punctually for weekly appointments but avoided eye contact and said very little. Due to her suicidal ideation she had been admitted to numerous mental health facilities.

Hanna Q is described as highly intelligent, and religious, with a refined appreciation of culture. She scored 62 on the Dissociative Experiences Scale (Bernstein-Carlson & Putnam, 1993) at the time of her initial assessment, indicating she was dissociative 62 percent of the time. Hanna Q could communicate that she had been abused when she was first referred, but it took well over a year before she was able, with difficulty and hesitancy, to begin to address specific aspects of the abuse she had suffered. Her father was highly regimented and emotionally distant, rarely speaking to his daughter unless to express anger. Hanna Q remarked, 'If you asked anyone about our family they'd say we were a perfect family. I didn't have bruises or scars for people to see'.

Pillars of society, her parents were never aware that as a young girl Hanna was being sexually abused by a church-affiliated family friend. When her mother saw ongoing evidence of sexually inappropriate behaviour by a relative towards Hanna Q, she rationalised it away rather than intervening to stop it. Hanna Q experienced multiple internalised voices of alter personalities. She exhibited no evidence of thought disorder or delusional thinking. Hanna had a personality state that was able to give detailed and even mundane information relating to the same day of the previous year: where she was, what she had for breakfast or lunch, and so on. Her capacity for eidetic memory was prodigious. She also had another alter personality that experienced life as if living one year in the future, which she believed was because she needed to know what was coming. Hanna Q had a substantial history of cutting and burning herself to cope with the repetitive denigrating statements from her negative alters as well as the intense discomfort she felt when experiencing depersonalisation. When Hanna's formal therapy came to a mutually planned conclusion six and a half years later, she had completed an undergraduate degree with a grade point average of 6.8 and was moving interstate to further her training.

(Adapted from Middleton, 2004a)

## QUESTIONS

1. Hanna Q saw a therapist for a whole year without saying much at all. What might be the reason for this?
2. Why do you think it took so long before she was able to give any detail of her traumas?

## Introduction

In this chapter, we discuss the dissociative disorders and the somatic symptom and related disorders. We cover these disorders together because both are associated with stressful and traumatic experiences. In the dissociative disorders, the person experiences disruptions of consciousness — he or she loses track of self-awareness, memory and identity. In the somatic symptom and related disorders, the person complains of bodily symptoms that suggest a physical defect or dysfunction, sometimes dramatic in nature. For some of the somatic symptom and related disorders, no physiological basis can be found, and for others, the psychological reaction to the symptoms appears excessive.

Dissociative disorders and somatic symptom and related disorders tend to be comorbid. Patients with dissociative disorders often meet the criteria for somatic symptom and related disorders, and vice versa; those with somatic symptom and related disorders are more likely than those in the general population to meet the diagnostic criteria for dissociative disorders (Brown, Cardena, Nijenhuis, Sar, & Van der Hart, 2007; Dell, 2006).

## 5.1 Dissociative disorders

**LEARNING OUTCOME 5.1** Summarise the symptoms, aetiological models and available treatments for dissociative disorders.

The DSM-5 includes four **dissociative disorders**: depersonalisation/derealisation disorder, dissociative amnesia, dissociative identity disorder (formerly known as multiple personality disorder) and other specified dissociative disorder. Table 5.1 summarises the key clinical features of the DSM-5 dissociative disorders.

**TABLE 5.1** Diagnoses of dissociative disorders

DSM-5 Diagnosis	Description
Depersonalisation/derealisation disorder	Persistent or recurrent experiences of unreality or detachment from one's mind, self or body; and/or alteration in the experience of the self and reality.
Dissociative amnesia	Inability to recall autobiographical information. This amnesia may be localised to an event or period of time, to a specific aspect of an event, or generalised to identity and life history.
Dissociative identity disorder (DID)	The presence of two or more distinct personality states or an experience of possession alongside recurrent episodes of amnesia.
Other specified dissociative disorder	Subclinical presentation of DID; dissociative states resulting from brainwashing or thought reform; mixed dissociative symptoms; dissociative trance, dissociative stupor or coma, and Ganser's syndrome.

Dissociative disorders are frequently found in the aftermath of trauma and are characterised by a disruption and discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control and behaviour (APA, 2013). Dissociation, the core feature of each dissociative disorder, involves some aspect of emotion, memory or experience being inaccessible consciously. Because dissociation is such a broad term (Allen, 2001), it is important to consider some of the different types of experience it encompasses.

Some types of dissociation are common. Many of you will have had experiences of studying so hard for a test that you lost track of time and might not even have noticed people coming and going from the room where you were studying. Some of you may have missed a turn on the road home when thinking about problems. These types of dissociative experiences are usually a harmless sign that one has been so focused on some aspect of experience that other aspects of experience are lost from awareness.

In contrast to common dissociative experiences like these, dissociative disorders are defined by more severe types of dissociation. Depersonalisation/derealisation involves a form of dissociation involving detachment, in which the person feels removed from the sense of self and surroundings. The person may feel 'spaced out', numb or as though in a dream (Holmes et al., 2005). Dissociative amnesia and dissociative identity disorder involve a more dramatic form of dissociation, in which the person cannot access important aspects of memory. In dissociative identity disorder, the gaps in memory are so extensive that the person loses all sense of having a unified identity.

A dissociative disorder is likely to be present if a person is having dissociative experiences 30 percent of the time as measured by the Dissociative Experiences Scale (Bernstein-Carlson & Putnam, 1993). Dissociative experiences, both normal and pathological, are very common among university students. On average, university students across the globe are experiencing dissociation 16.6 percent of the time, although the rate for Australian students — 12.4 percent — is lower (Kate, 2017). Furthermore, 7.5 percent of university students have been found to meet the criteria for a dissociative disorder at some point in their life (Nilsen, 2000).

### Why does dissociation occur? Exploring different perspectives

There are two leading accounts of ongoing dissociative symptoms in the current literature: the trauma model and the fantasy model. The **trauma model** is based on the premise that dissociation is a reaction to traumatic stress or severe psychological adversity. Trauma model theorists are of the view that ongoing dissociative symptoms are a consequence, if not a continuation, of previous responses to physical and sexual abuse and assault, emotional abuse and neglect, physical neglect, and other severe stress or trauma such as witnessing violence, which is supported by numerous studies (see overview in Dalenber et al., 2014). Contemporary attachment researchers have shown dissociation also occurs when a potentially protective caregiver is perceived as a threat or consistently fails to respond adequately to the young child's attachment bids and the child is faced with a conflict between two incompatible behaviours: seeking and avoiding proximity to the attachment figure (Carlson, Cicchetti, Barnett, & Braunwald, 1989; Lyons-Ruth, 2003; Main & Morgan, 1996; Ogawa, Sroufe, Weinfield, Carlson, & Egeland, 1997; Siegel, 2014; Main & Hesse, 1990). Australian research demonstrates that dissociation is most likely to occur in response to a trauma if the child is also living in a negative family environment, including where there is a lack of emotional support (Irwin, 1996) and where the caregivers failed to protect the child, blamed the child or enabled, incited or perpetrated the mistreatment (Kate, 2016a).

The concept of dissociation as a form of post-traumatic response is shared by many prominent writers on trauma. van der Kolk (2014, p. 66) calls dissociation the 'essence of trauma', holding that during trauma, overwhelming experience is split off from immediate high-level cognitive and affective processes as an adaptive response. Later, in pathological dissociation, the emotions, sounds, images, thoughts and physical sensation related to the trauma intrude into the present as psychological fragments. In its most extreme form, this pathological splitting may result in the emergence of distinct personal identities (van der Kolk, 2014, p. 278). Perry (2003) explains that dissociation is a primary adaptive response to threat most common in younger children during traumatic events that are characterised by an inability to escape, which is supported by Porges's (2011, 2013) polyvagal theory, which posits that the body's parasympathetic defence response of immobilisation (i.e., the freeze response) is initiated when a person is unable to fight or flee from danger and this produces behavioural shutdown, which includes dissociation.

Most researchers who reject the trauma model adopt some form of **fantasy model**, which postulates that dissociative disorders do not primarily arise from trauma, but occur as a result of fantasy proneness, suggestibility and cognitive failures. The fantasy model is based on the premise that fantasisers may confuse imagined events with factual autobiographical memories and adopt a more liberal response criterion for reporting an experience as genuine (Giesbrecht, Lynn, Lilienfeld, & Merckelbach, 2008) and that this propensity to fantasise, coupled with suggestive therapeutic procedures and efforts to recover memories, may lead the individual to attribute distinctive, but false, memories to the genesis of a dissociative disorder (Lynn, Lilienfeld, Merckelbach, Giesbrecht, & van der Kloet, 2012). More recently, fantasy model theorists (Lynn et al. 2014) have conceded that 'trauma may sometimes play an aetiological role in dissociation', although they state this is because traumatised individuals are more likely to fantasise, have disrupted sleep, be suggestible and therefore likely to incorrectly think they have a dissociative disorder, while in reality they are just enacting the role of someone who is dissociative due to their exposure to sociocultural influences (e.g., TV, books) and the influence of overzealous therapists.

A link between fantasy proneness and dissociative symptoms, however, is not inconsistent with the trauma model. Vaillant (2011) categorises both dissociation and fantasy as defence mechanisms. Winnicott (1971), who also considered fantasy to be a defence mechanism, explains that fantasy often operates in tandem with dissociation as a dissociated state provides the backdrop necessary for pleasurable imaginings to take precedence over reality in order to provide respite from harsh physical and psychological experiences. The use of fantasy as a coping mechanism is recognised by Lynn and Rhue (1988) who found that fantasy-prone university students were more likely than their peers to have abusive or lonely childhoods and the authors suggest that those fantasisers who received frequent and severe parental punishment used their imaginative capacity to minimise physical and psychic pain and to preserve a relatively positive view of the abusive environment. While fantasy proneness and dissociation are correlated, strong empirical evidence demonstrates dissociation remains related to trauma history after controlling for fantasy proneness (Dalenber et al., 2012).

While poor sleep, suggestibility and therapist suggestion may play a small role in dissociative disorders, none of these factors adequately explain the widespread phenomenon of pathological dissociation. First, a link between traumatic experience and disrupted sleep is in itself uncontroversial and poor sleep is known to have a negative impact on mental health more generally. However, sleep studies do not support the fantasy model notion that disrupted sleep leads to pathological levels of dissociation; rather, these indicate dissociation precedes or co-occurs with a disruption to the sleep–wake boundaries as dissociation is specifically related to unusual sleep experiences, which include narcolepsy, REM sleep without muscle weakness and sleepwalking (Koffel & Watson, 2009). Second, the link between dissociation and suggestibility proposed by fantasy model theorists was found to be negligible in a meta-analysis, with dissociation predicting only 1 to 3 percent of suggestibility (Brand et al., 2016). And third, there is compelling evidence that dissociative disorders have been diagnosed where suggestibility played no role in the onset of the disorder. For example, a review of studies of psychiatric inpatients in the Netherlands, Norway, the United States, Canada and Turkey (Ross, Duffy, & Ellason, 2002) found 16 percent of the patients met the criteria for a dissociative disorder. Subjects were not claiming to have a dissociative disorder, had never received a dissociative disorder diagnosis and had never received treatment for a dissociative disorder. In sum, the hypothesis that dissociation renders individuals prone to fantasy and subsequently leads to false memories of trauma has no robust empirical support (Staniloiu & Markowitsch, 2014).

#### QUESTIONS

1. Summarise the role fantasy proneness plays in both the fantasy and trauma models of dissociation.
2. Which model of dissociation do you think provides the most convincing explanation of dissociation? Why?

The prevalence of dissociative disorders is difficult to accurately estimate as it is understudied. Only two studies have assessed the lifetime prevalence of the dissociative disorders in community samples using face-to-face diagnostic interviews with all participants — a Canadian study (Ross, 1991) found a rate of 13 percent and a Turkish all-female study (Sar, Akyuz, & Dogan, 2007) found a rate of 18 percent. The studies indicate that dissociative disorders are quite common. Both studies relied on the Dissociative Disorders Interview Schedule (Ross et al., 1989), which has been found to over-diagnose dissociative amnesia and under-diagnose depersonalisation disorder (Ross, Duffy, & Ellason, 2002) when compared to the Structured Clinical Interview for DSM-IV Dissociative Disorders (SCID-D) (Steinberg, 1994), which is considered the gold standard in diagnosing dissociative disorders (Welburn et al., 2003). A study conducted in New York State (Johnson, Cohen, Chen, Casen, & Brook, 2006) found the rate of dissociative disorders to be 8.6 percent using the SCID-D; however, this lower rate may be attributable to pre-screening with the Dissociative Experiences Scale (Waller, Putnam, & Carlson, 1996), which is not adept at diagnosing dissociative amnesia or depersonalisation/derealisation disorder (Nilsen, 2000; Simeon, 2009).

## Depersonalisation/derealisation disorder

As described above, **depersonalisation/derealisation disorder** involves a disconcerting and disruptive sense of detachment from one's self or surroundings. Depersonalisation is defined by a sense of being detached from one's self (e.g., being an observer outside of one's body). Derealisation is defined by a sense of detachment from one's surroundings, such that the surroundings seem unreal (Heller et al., 2013). People with this disorder may have the impression that they are outside their bodies, viewing themselves from a distance or looking at the world through a fog. Sometimes they feel mechanical, as though they are robots.

About 2.5 percent of people met the lifetime diagnostic criteria for depersonalisation/derealisation (Ross, 1991; Sar et al., 2007) and it occurs equally in men and women (Simeon, Knutelska, Nelson, & Guralnik, 2003). More than a third of university students (Hunter, Sierra, & David, 2004) and 23 percent

of the general population (Aderibigbe, Bloch, & Walker, 2001) report that they have experienced at least fleeting moments of depersonalisation or derealisation in the past year, but these mild and intermittent symptoms are rarely of concern. The DSM-5 criteria for depersonalisation/derealisation disorder specify that the symptoms must be persistent or recurrent.

Amnesia is not part of the DSM-5 diagnostic criteria for depersonalisation/derealisation disorder. However, research indicates the disorder is accompanied by varying degrees of amnesia; for example, many with the condition persistently complain of having a poor memory, although they do not lose time (Simeon, 2009).

#### DSM-5

##### **DSM-5 criteria for depersonalisation/derealisation disorder**

- People with depersonalisation disorder experience detachment from their mental processes or body, as though they are in a dream.
- People with derealisation disorder have experiences of unreality of surroundings.
- Symptoms are persistent or recurrent.
- Reality testing remains intact.
- Symptoms are not explained by substances, another dissociative disorder, another psychological disorder or by a medical condition.

The symptoms of depersonalisation and derealisation are usually triggered by stress. Depersonalisation/derealisation disorder usually begins in adolescence and it can start either abruptly or more insidiously. Most people who experience depersonalisation also experience derealisation and the course of symptoms is similar for both symptoms. Symptoms are often continuously present for years (Simeon, 2009). As in the case of Mrs A, described below, chronic childhood trauma is often reported, including physical and sexual abuse, but especially emotional abuse (Simeon, Guralnik, Schmeidler, Sirof, & Knutelska, 2001). Later life stressors can also trigger depersonalisation, for example, the death or suicide of someone close, as well as romantic, educational or occupational struggles (Simeon et al., 2003). Depersonalisation is also relatively common during panic attacks (Marquez, Segui, Garcia, Canet, & Ortiz, 2001) and from using marijuana, hallucinogens, 3,4-Methylenedioxymethamphetamine (MDMA) or ketamine (Simeon, 2009).

Comorbid personality disorders are frequent, and during their lifetime, about 90 percent of people with this disorder will experience anxiety disorders and depression (Simeon et al., 2003). The DSM-5 diagnostic criteria for depersonalisation/derealisation disorder specify that the symptoms can co-occur with other disorders but should not be entirely explained by those disorders. It is important to rule out disorders that commonly involve these symptoms, including schizophrenia, post-traumatic stress disorder and borderline personality disorder (Maldonado, Butler, & Spiegel, 1998).

Using the Dissociative Experiences Scale (DES), the recommended cut-off score is 30 for identifying those who might have a dissociative disorder (Bernstein-Carlson & Putnam, 1993). Yet the average score for people diagnosed with depersonalisation disorder is 27 and for derealisation disorder is 15 (Simeon, 2009), the latter being slightly less than in the average university student. To avoid this problem, the depersonalisation scale of the DES can be used as a stand-alone measure (Simeon, 2009) or a more specific instrument such as the Cambridge Depersonalisation Scale (Sierra & Berrios, 2000) can be used.

Depersonalisation/derealisation disorder remains a poorly understood and under-researched condition. Psychotherapy case reports have indicated that psychodynamic psychotherapy and hypnosis-based treatment, combined with eye movement desensitisation and reprocessing (EMDR), may be useful (Somer, Amos-Williams, & Stein, 2013).

## CLINICAL CASE

### Mrs A

Mrs A was a 43-year-old woman who lived with her mother and son, and worked in a clerical job. She had experienced symptoms of depersonalisation several times per year for as long as she could remember. 'It's as if the real me is taken out and put on a shelf or stored somewhere inside of me. Whatever makes me *me* is not there. It is like an opaque curtain ... like going through the motions and having to exert discipline to keep the unit together.' She had found these symptoms to be extremely distressing. She had experienced panic attacks for one year when she was 35. She described a childhood trauma history that included nightly genital fondling and frequent enemas by her mother from her earliest memory to age 10. (Simeon et al., 1997, p. 1109)

### QUESTIONS

1. Do you think Mrs A is more likely to have depersonalisation disorder or derealisation disorder? Why?
2. Mrs A says, 'It's as if the real me is taken out and put on a shelf or stored somewhere inside of me'. Could this indicate she might have a more severe dissociative disorder or is this consistent with depersonalisation/derealisation disorder?

## Dissociative amnesia

The person with **dissociative amnesia** is unable to recall important personal information, usually information about some traumatic experience. The holes in memory are too extensive to be explained by ordinary forgetfulness. The information is not permanently lost, but it cannot be retrieved during the episode of amnesia, which may last as briefly as minutes or for decades. Individuals may report multiple episodes of dissociative amnesia and a single episode may predispose the individual to future episodes. Some episodes of dissociative amnesia resolve rapidly, for example, when the person is removed from combat or some other stressful situation, whereas other episodes persist for long periods of time. Some individuals may gradually recall the dissociated memories over a period of years and those with chronic amnesia may retain a very impaired autobiographical memory (APA, 2013).

Most often the memory loss of amnesia involves information about some part of a traumatic experience, such as witnessing the sudden death of a loved one, war service or experiencing a natural disaster, or severe stress, such as marital discord, personal rejection, or financial or occupational difficulties. Nonetheless, not all amnesias immediately follow trauma (Hacking, 1998). During the period of amnesia the person's behaviour may or may not appear to be acutely symptomatic, although the memory loss may cause some disorientation. Procedural memory remains intact — the person remembers how to answer the phone, ride a bike and execute other complex actions, even though he or she is unable to remember specific events.

## DSM-5

### DSM-5 criteria for dissociative amnesia

- People with dissociative amnesia have an inability to remember important autobiographical information, usually of a traumatic or stressful nature, that is too extensive to be ordinary forgetfulness.
- The amnesia is not explained by substances or by other medical or psychological conditions.
- The person has a dissociative fugue subtype if the amnesia is associated with apparently purposeful travel or bewildered wandering that is associated with amnesia for identity or for other important autobiographical information.

In a more severe subtype of amnesia called **fugue** (from the Latin *fugere*, ‘to flee’), the memory loss is more extensive. The person typically disappears from home and work. Some may wander away from home in a bewildered manner. Others may take on a new name, a new home, a new job and even a new set of personality characteristics. The person may even succeed in establishing a fairly complex social life. Fugue is rare even among people with dissociative amnesia, but is commonly seen in individuals with dissociative identity disorder (APA, 2013).

Dissociative amnesia raises fundamental questions about how memory works under stress. Psychodynamic theory suggests that in dissociative amnesia, traumatic events are repressed, dissociated from consciousness. In this model, memories are forgotten because they are so aversive. Dissociative amnesia involves unusual ways of responding to stress. For example, extremely high levels of stress hormones could interfere with memory formation (Andreano & Cahill, 2006) and chronic stress, such as repeated abuse, might have more detrimental effects on memory than acute stress (Reinhold & Markowitsch, 2009). Dissociative amnesia is based on the premise that in the face of severe trauma, memories may be stored in such a way that they are not accessible to awareness later when the person has returned to a more normal state (Kihlstrom, Tataryn, & Holt, 1993). This ‘state dependent retrieval’ of fear-based memories has been confirmed in animal studies (Jovasevic et al., 2015) and experimental evidence also demonstrates that dissociative states affect memory in humans (Brewin, Ma, & Colson, 2013).

## FOCUS ON DISCOVERY 5.2

### Debates about repression: recovered memories of abuse in childhood

While the concept of recovered memory has been a focus for debate, particularly since the 90s, evidence demonstrates that memories of trauma can be lost and found, continuous memories can be distorted and, under certain conditions, false memories can be created. Cognitive researchers have demonstrated that all memory, not simply recovered memory, is reconstructive and should be examined equally closely (Dalenberg et al., 2014).

#### Memories can be recovered

Hundreds of scientific publications spanning well over a century document how the memory of traumatic events, including natural disasters, accidents, war trauma, kidnapping, torture, concentration camps, physical abuse and sexual abuse in childhood, can be lost only to resurface years later (Middleton, Di Marni Cromer, & Freyd, 2005; van der Kolk, 2014). Around 15 percent of people who have experienced any kind of traumatic event report a period of time when they had no memory of it, with the rate being significantly higher for interpersonal trauma (Elliott, 1997). The risk of a person being unable to remember large parts of their childhood after age four is increased 4.4-fold if they experienced four or more different types of adverse childhood experiences (see focus on discovery 5.3 for more information). The deficits in memory in individuals with histories of chronic childhood trauma are attributed to neurobiological defects resulting from the trauma, which is apparent in the reduced size of the hippocampus and amygdala — the areas of the brain responsible for memory storage and retrieval (Anda et al., 2006). Although studies examining recovery of traumatic memory may use terms such as amnesia, repression or dissociation, the cognitive process or processes by which the memory appears to be lost and then recovered remains elusive.

People are more likely to report forgetting sexual abuse than other types of trauma. Studies in the general population indicate that between 20 and 32 percent of individuals reporting sexual abuse state there was a time when they could not recall it had occurred (Edwards, Fivush, Anda, Felitti, & Nordenberg, 2001; Elliott, 1997; Scheflin & Brown, 1996; Wilsnack, Wilsnack, Wonderlich, Kristjanson, & Vogeltanz-Holm, 2002). A study with Australian university students (Kate, 2016b) found half of the respondents did not have a clear memory of the sexual abuse, which included 9 percent who had experienced a time when they could not remember it at all and still only had a partial memory, as well as 2 percent who had no memory of it at all but had been informed the abuse occurred. Memory problems could not be accounted for by therapist suggestion, drugs or alcohol, infantile amnesia or sociocultural memory triggers such as books, films or other media.

As sexual abuse is nearly always clandestine and there is rarely forensic evidence years later, nearly all studies on recovered memories of abuse rely on self-reports by participants. Williams' (1994, 1995) landmark study followed 136 women who, before the age of 12, were admitted to a hospital's emergency department for treatment and forensic evidence collection following sexual abuse. Of the 80 women who recounted the documented abuse experience to the interviewer, when asked if they had been sexually abused, 12 (16 percent) reported that there was a time when they did not remember that this had happened to them. However, it is likely that the rate of recovered memory is far higher as an additional 49 women (38 percent of the total sample) did not disclose the documented abuse at all. This finding was unable to be explained by infantile amnesia. For some women this may have been a reluctance to disclose, but most who did not disclose the abuse that took them to the emergency room told the interviewers embarrassing, upsetting and stigmatising personal experiences, including other incidents of sexual abuse. The Williams study clearly shows that memories of documented sexual abuse can be lost and recovered.

An inability to recall childhood abuse tends to be more common where the abuse is especially violent, where the child fears for their safety if the abuse was disclosed to others (Briere & Conte, 1993; Elliott & Briere, 1995; Herman & Schatzow, 1987) and where the perpetrator is a relative, particularly if they are the child's caregiver (Duggal & Sroufe, 1998; Freyd, DePrince, & Zurbriggen, 2001; Herman & Schatzow, 1987; Moormann, Albach, & Bermond, 2012). Freyd (1996) presents the concept of 'betrayal trauma' to describe the logic of 'forgetting' abuse inflicted by someone whom the child is dependent upon.

Are recovered memories accurate? Overall, research with abused, non-abused, clinical, non-clinical and experimental participants finds that recovered memories and continuous memories are equally accurate and are more likely to be true than false (Dalenberg, 2006). No studies have yet demonstrated that all or most of the recovered memories were false, nor has any study in which evidence of trauma was systematically sought found an absence of corroboration in most recovered memory clients (Dalenberg et al., 2014). Trauma experts agree that recollections of child abuse experiences may at times mix actual events with fantasy, confabulated details, abusers' rationalisations of the events or condensations of several events (ISSTD, 2011).

The view that traumatic memory can be lost and recovered is supported by professional organisations around the world. The Australian and New Zealand College of Psychiatrists (RANZCP) emphasised over a decade ago the extent of physical and sexual abuse of children, the harmful nature of such abuses and that 'memory of such abusive experiences may be absent for considerable and varied periods of life and may be recalled under any of a variety of circumstance' (Middleton et al., 2005).

### **False memories can be implanted**

Memories of abuse may be patchy, distorted and contain inaccuracies, but is it possible memories of abuse can be completely untrue? False memories of traumatic events have been created in laboratory situations. Porter, Yuille and Lehman (1999) gave university students details of two events — one true and one false. The false event, which included experiences such as being lost, harmed by another child or attacked by an animal, was introduced by the researcher as being true according to the student's parents. When the student was unable to remember the false event, they were asked to imagine the event and told 'most people are able to retrieve lost memories if they try hard enough'. The interviewer used guided imagery to encourage memories to surface and instructed the student to 'visualise what it might have been like and the memory will probably come back to you'. The student was encouraged to take a few minutes each night to try to recover the memory and write down any thoughts pertaining to it. The process was repeated in two further interviews to imbed the false memory, including writing a report about the memory. At the end of this process 26 percent of students 'recovered' a complete memory for the false event and another 30 percent exhibited a partial false memory demonstrating that believed-in, plausible memories can be implanted in a laboratory setting. However, the research also supports the view that memories of real events can be lost, with 12 percent of participants having no memory of the genuine stressful or frightening event described by their parents.

Although the trait of suggestibility only predicts 3 percent or less of dissociative tendencies, dissociative individuals may be at risk of a false memory if they are directly or indirectly encouraged to imagine and ruminate over a plausible, believed-in event (Dalenberg et al., 2012).

Studies of memory show that recall of even a major event such as the terrorist attacks on 9/11 can be considerably distorted.



As all memory is fallible, it is recommended that clinicians adopt a respectful neutral stance on memories that are presented, avoid suggestive and leading techniques and provide ongoing discussion and education about the nature of memory (ISSTD, 2011). Social scientists and the courts share a heavy responsibility in deciding whether a given recovered memory is an accurate reflection of a criminal event. Erring in either direction could result in an injustice to either the accuser or the accused.

### QUESTIONS

1. Do you think there is a difference in a person accepting a memory of a fairly common childhood trauma such as being bitten by a stray dog, compared to a memory of being sexually abused by their favourite uncle? Why?
2. In the two examples in question 1, can you see any 'advantages' in forgetting? If so, are these advantages similar or different for each example?

Dissociative amnesia is a common experience with a prevalence of 7.5 percent found in general population studies (Ross, 1991; Sar et al., 2007). A prevalence rate of 1.8 percent was found in an American study using the Structured Clinical Interview for Dissociative Disorders (Johnson et al., 2006), but this may be attributed to pre-screening being conducted with the Dissociative Experiences Scale, which is not effective in detecting dissociative amnesia. The Steinberg Dissociative Amnesia Questionnaire (Steinberg & Schnall, 2001) has been found to be a reliable tool in screening for the disorder (Sar, Akyuz, Kundakci, Kiziltan, & Dogan, 2004).

At present, no evidence-based treatments are available for dissociative amnesia. Psychotherapeutic treatment in dissociative amnesia usually follows a phasic approach, which initially focuses on stabilisation, securing patient safety and symptom reduction, and then moves on to processing the traumatic events or psychological conflicts (Staniloiu & Markowitsch, 2014). As the amnesia remits, there may be considerable distress, suicidal behaviour and symptoms of post-traumatic stress disorder (PTSD).

In diagnosing dissociative amnesia, it is important to rule out other common causes of memory loss, such as dementia, substance abuse and medication side effects. Heavy drinking can cause blackouts and medications such as the benzodiazepines used in the treatment of anxiety (e.g., Valium) and the sedative-hypnotics used in the treatment of insomnia (e.g., Ambien) can cause amnesia, and so a key diagnostic consideration is whether any head injury, medication use or substance use might have preceded the episode. Dementia can be fairly easily distinguished from dissociative amnesia. In dementia, memory fails slowly over time, is not linked to stress and is accompanied by other cognitive deficits, such as an inability to learn new information.

### CLINICAL CASE

#### AJW

AJW was taken from her Indigenous mother and white father when she was 2 years of age and placed in a children's home where she lived until she was 12 when the home closed down. AJW was subjected to physical beatings and was sexually abused by one of the male 'house parents' of the home. At the age of eight, AJW went to court in relation to the widespread sexual abuse that was occurring in the children's home, but she recounts that she was too petrified to give evidence, as her house parent was in the courtroom. AJW explains that for many years she had no recollection of any sexual abuse occurring. When AJW was around 26 years old, she was admitted to hospital for an operation and saw a document stating that she had been sexually abused as a child and that, upon turning 18, she must be told she was sexually molested as a child. AJW said that before reading the report she had no memory of the abuse. Evidence pertaining to AJW's case is documented by the Royal Commission into Institutional Responses to Child Sexual Abuse (2015).

The dormitory of the children's home where AJW lived



#### QUESTIONS

1. Does AJW's case provide support for the trauma model or the fantasy model of dissociation? Why?
2. What circumstances may have made AJW more at risk of forgetting the sexual abuse she had experienced?

## Dissociative identity disorder

The diagnosis of **dissociative identity disorder (DID)**, formerly labelled multiple personality disorder, requires that a person have at least two separate personalities or alters — different modes of being, thinking, feeling and acting that exist independently of one another and that emerge at different times. Each determines the person's nature and activities when it is in command. The primary alter may be totally unaware that any other alter exists and may have no memory of what those other alters do and experience when they are in control.

Case reports suggest that there is typically one primary personality and that this is usually the alter that seeks treatment. Most commonly, two to four alters are identified when the diagnosis is made, but over the course of treatment others may emerge. Each alter may be quite complex, with its own behaviour patterns, memories and relationships. Usually, the personalities of the different alters are quite different from one another, sometimes seemingly polar opposites. Case reports have described alters who have different handedness, wear glasses with different prescriptions, like different foods and have allergies to different substances. The alters are aware of lost periods of time, and the voices of the others may intrude (often loudly) into an alter's consciousness, even though the alter may not know to whom these voices belong.

In some cultures, alters may be understood as spirits who take control of the person's body, and these experiences of possession that are part of broadly accepted spiritual or cultural practice are not a recognised form of DID. However, the DSM-5 extended the diagnosis of DID to include possession states that are involuntary, distressing, uncontrollable and often recurrent or persistent; involve conflict between the individual and his or her surrounding family, social or work milieu; and are manifested at times and in places that violate the norms of the culture or religion.

DID is generally considered to be rare, yet studies demonstrate that between 1 and 3 percent of the general population will meet the diagnostic criteria (Ross, 1991; Johnson et al., 2006; Sar et al., 2007). International studies have found the prevalence of DID in inpatient and outpatient psychiatric settings to be approximately 5 percent.

### DSM-5

#### DSM-5 criteria for dissociative identity disorder (DID)

- People with DID have a disruption of identity characterised by two or more distinct personality states (alters) or an experience of possession. These disruptions lead to discontinuities in the sense of self or agency, as reflected in altered cognition, behaviour, affect, perceptions, consciousness, memories or sensory-motor functioning. This disruption may be observed by others or reported by the patient.
- People with DID have recurrent gaps in memory for events or important personal information that are beyond ordinary forgetting.
- Symptoms are not part of a broadly accepted cultural or religious practice.
- Symptoms are not due to drugs or a medical condition.
- In children, symptoms are not better explained by an imaginary playmate or by fantasy play.

DID is rarely diagnosed until adulthood, but often after their diagnosis, patients will recall symptoms dating back to childhood. It is more severe and extensive than the other dissociative disorders (Mueller-Pfeiffer et al., 2012). DID is much more commonly diagnosed in women than in men. Other diagnoses are often present, including post-traumatic stress disorder, major depressive disorder and somatic symptom disorders (Middleton & Butler, 1998; Rodewald, Wilhelm-Goling, Emrich, Reddemann, & Gast, 2011). Personality disorders are also common (Johnson et al., 2006). DID is commonly accompanied by other symptoms such as headaches, hallucinations, suicide attempts and self-injurious behaviour, as well as by other dissociative symptoms such as amnesia and depersonalisation (Scroppo, Drob, Weinberger, & Eagle, 1998).

### Aetiology of DID

Two major theories have been advanced for DID: the trauma model and the fantasy model. Both theories suggest that severe physical or sexual abuse during childhood sets the stage for DID. Almost all patients

in therapy for DID report severe childhood abuse (Middleton & Butler, 1998; Dalenberg et al., 2012). Autobiographical amnesia, which may span years of childhood, is a common feature of DID so childhood abuse cannot be ruled out even if no such history has been disclosed. Since few people who are abused develop DID, both models focus on why some people do develop DID after abuse. As we will see, considerable debate has arisen between the proponents of these two approaches.

### **The trauma model**

Large-scale clinical and epidemiological studies in Australia, Turkey, Puerto Rico and North America have found that DID is linked to severe, chronic abusive and traumatic experiences in childhood, typically at the hands of a caregiver (see Dorahy et al., 2014). Several studies using corroborating documentation from hospital, police and child protection agencies confirm histories of severe abuse in DID (Dorahy et al., 2014). Sexual abuse starting at a young age is extremely common with the average duration being 15 years and involving three perpetrators, often including one or both caregivers (Ross & Ness, 2010). In a detailed report on ten women with DID where incestuous abuse had continued on into adulthood, the average duration of abuse was 31 years and their abuse experiences were congruous with multiple reports in the international press (Middleton, 2013; Middleton, 2014).

The trauma model proposes that some people are particularly likely to use dissociation to cope with trauma and this is seen as a key factor in causing people to develop alters after trauma (Gleaves, 1996). Research supports four important tenets of this model. First, there is evidence that children who are abused are at risk for developing dissociative symptoms (Chu, Frey, Ganzel, & Matthews, 2000). Second, there is evidence that children who dissociate are more likely to develop psychological symptoms after trauma (Kisiel & Lyons, 2001). Third, evidence of clinical symptoms of DID are often found to be present when neither the clinician nor the patient have considered a dissociative disorder diagnosis (see clinical case below). Fourth, differences in brain activity can be witnessed between the different personality states of people with DID using fMRI, however these differences are not evident in people role-playing the disorder (Schlumpf et al., 2014).

### **The fantasy model**

According to the fantasy model, people who have been abused seek explanations for their symptoms and distress and alters appear in response to suggestions by therapists, exposure to media reports of DID or other cultural influences (Lilienfeld et al., 1999; Spanos, 1994). This model, then, implies that DID could be **iatrogenic** (created as a result of treatment) in that the person often learns to role-play these symptoms within treatment. This does not mean, however, that DID is viewed as conscious deception; the issue is not whether DID is real but how it develops.

In the early 1990s, many of the treatment manuals for DID recommended therapeutic techniques that reinforce clients' identification of different alters, such as interviewing clients about their identities after administering hypnosis or sodium pentothal (Nathan, 2011; Ross, 1991). It was suggested that

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Physical or sexual abuse in childhood is regarded as a major factor in the development of dissociative disorders.



reinforcement and suggestive techniques might promote false memories and DID symptoms in vulnerable people (Lilienfeld et al., 1999).

The famous case of Sybil is often cited as an example of how a therapist might elicit and reinforce stories of alters. It has been claimed that in Sybil's case, her 16 alters were created by a therapist who gave substance to Sybil's different emotional states by giving them names and who helped Sybil elaborate on her early childhood experiences while administering sodium pentothal, a drug that has been shown to contribute to false memories (Borch-Jacobsen, 1997; Nathan, 2011). The controversy continued as other evidence, including a series of handwritten letters between Sybil and a close friend that were made public in 2013 (Preston, 2013), supports the diagnosis of DID.

We will never have experimental evidence for the fantasy model, since it would be unethical to intentionally reinforce dissociative symptoms. Given this reality, what kinds of evidence have been raised in support of the fantasy model? While rare, cases of iatrogenic DID have been described in North America. The common features of these cases are malpractice by the therapist; the person becoming an inpatient of a psychiatric hospital for one to two years and being placed on a ward with other DID patients; extensive use of hypnosis and psychotropic medication; positive feedback given for 'recovering' memories of trauma; and phone calls and visits restricted or banned so the person is isolated from family and friends (Ross, 1997). While it seems possible to create a condition akin to DID iatrogenically, these findings suggest it is more than a matter of therapist suggestion, but requires intensive, long-term and coercive interventions in a particularly vulnerable person. Individuals with DID are not more fantasy prone than the general population, a finding that refutes the idea that DID is caused by fantasy proneness or suggestibility (Reinders, Willemsen, Vos, den Boer, & Nijenhuis, 2012).

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In the film version of *Sybil*, about a famous case of dissociative identity disorder, the title role was played by Sally Field.



### **What explains the increase in the number of DID cases since the 1970s?**

Although descriptions of anxiety, depression and psychosis have abounded in literature since ancient times, there were almost no identified reports of DID before 1800 (Pope, Poliakoff, Parker, Boynes, & Hudson, 2006). The earliest recorded DID case is Jeanne Fery in the sixteenth century (van der Hart, Lierens, & Goodwin, 1996) and reports of DID were relatively frequent between 1890 and 1920, with 77 case reports appearing in the literature during that time (Sutcliffe & Jones, 1962). After 1920, reports of DID declined until the 1970s, when they increased markedly, not only in the United States but also in countries such as Japan (Uchinuma & Sekine, 2000). What explains the increase in DID diagnosis? There are two perspectives.

Fantasy model theorists suggest that popular culture is to explain for the rise in DID diagnoses. The case of Eve White, popularised in the book *The Three Faces of Eve* as well as a movie, provided a highly detailed report of DID in 1957. The popular 1973 book *Sybil* presented a dramatic case with 16 personalities (Schreiber, 1973). The book sold more than 6 million copies in the first four years in print and more than a fifth of all Americans watched a television adaptation broadcast in 1976 (Nathan, 2011). A series of other case reports were published in the 1970s as well. DSM-III, which appeared in 1980,

defined the diagnosis of DID for the first time (Putnam, 1996). Some critics hypothesise that the heightened professional and media attention to this diagnosis led some therapists to suggest strongly to clients that they had DID, sometimes using hypnosis to probe for alters, which created DID iatrogenically.

A view consistent with the trauma model is that DID is not as visible as other disorders and, without adequate tools or awareness, it remains undetected. It is known that individuals with DID rarely volunteer information about dissociative symptoms. This may be due to patients' early trauma and attachment difficulties and the resultant mistrust of others, especially authority figures; the fact that some individuals do not realise that having alters is not a normal experience (ISSTD, 2011); and embarrassment and confusion about their symptoms and a resulting desire to hide these from clinicians and others (APA, 2013). Unless clinicians know what to look for, the DID may be missed. The introduction of the DSM-III diagnostic criteria for DID in 1980 accompanied by the Dissociative Experience Scale in 1986 and the Structured Clinical Interview for Dissociation in 1994 provided tools for detection. For example, unstructured diagnostic interviews of more than 11 000 outpatients at a major psychiatric centre did not identify a single case of DID (Mezzich, Fabrega, Coffman, & Haley, 1989), yet using validated screening and diagnostic tools, DID has been found across the globe, including in Australia, the Philippines, Turkey, Puerto Rico, Scandinavia, Japan, Canada, Ireland, the UK, Argentina, Oman, China and Iran (Dorahy et al., 2014). The significant problem is that DID still most often goes unrecognised. Clinicians may view DID as a rare disorder with a florid and dramatic presentation (such as that portrayed by Toni Collette in the TV show *United States of Tara*), yet only a small minority present with visibly distinct alternate identities (ISSTD, 2011). The lower profile that dissociative disorders have compared to other diagnoses means that other co-existing conditions are more likely to be identified and treated and may be mistaken for a schizophrenia due to the similarity of symptoms such as hearing voices (Dorahy, et al., 2014).

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In the TV show *United States of Tara*, family members struggle as the mother of the family, played by the Australian actress Toni Collette, shifts between different alters.



## CLINICAL CASE

### Symptoms of dissociative identity disorder (DID) prior to diagnosis

In this example, we look at one young Australian woman's medical records compiled from psychiatric hospitals over a five-year period. During this time, a DID diagnosis was never considered by the woman or her therapists. Her records also confirm a history of severe incestuous childhood abuse, which resulted in her father being jailed. These are some excerpts from her medical records.

- Able to give a past history of behaviours suggestive of a borderline personality, identity diffusion, several relationship difficulties, periodic thoughts of self harm, ... affective instability and longstanding substance abuse
- Within 24 hrs she was describing experiencing hearing voices, both male and female, instructing her to hurt herself. She said that these had occurred periodically for many years
- Has an unusual and unclear story, but apparently she showed abusive behaviour ... and described visual hallucinations and auditory hallucinations
- By the time she had reached the [Royal St Elsewhere] she denied any memory for the events that had occurred at the Maternity Hospital
- Diagnosis was difficult in the setting of a changing story
- She also described a number of psychotic features such as thought broadcasting, auditory hallucinations and ideas of reference ... it was decided to recommence Electroconvulsive therapy
- On interview she was extremely distressed and used the word 'we' to describe herself. She felt that there were several people inside herself. There was a Sally, who was small and also ran away the whole time. There was Ruth, who was dirty and a tramp, Gary — the strong one and Annie who works with Gary
- She has no memories prior to the age of 12
- She also described visual hallucinations and olfactory and gustatory hallucinations ... She described symptoms of PTSD, awakening at night with vivid visions of her earlier sexual abuse
- During her admission, she had episodes of increased psychosis and suicidal ideation. There were periods when she appeared quite well and had several successful leaves. (Middleton, 2004b)

In the five-year period of frequent hospitalisation a dissociative disorder diagnosis was never considered.

### QUESTIONS

1. Which criteria for DID are present in this case?
2. What symptoms are similar to schizophrenia and how might you differentiate between the two disorders in this case?

## Treatment of DID

There is widespread agreement on several principles in the treatment of DID (Brand et al., 2012; ISSTD, 2011). These include the importance of an empathic and gentle stance, with the goal of helping the client function as one wholly integrated person. The goal of treatment should be to convince the person that splitting into different personalities is no longer necessary to deal with traumas. In addition, as DID is conceptualised as a means of escaping from severe stress, treatment can help teach the person more effective ways to cope with stress, such as emotion regulation strategies. Psychoeducation can help a person to understand why dissociation occurs and to begin to identify the triggers for dissociative responses in day-to-day life (Brand et al., 2012). Often, people with DID are hospitalised to help them avoid self-harm and to offer more intensive treatment. The International Society for the Study of Trauma and Dissociation outline a three-stage treatment approach of establishing safety, stabilisation and symptom reduction; confronting, working through and integrating traumatic memories; and identity integration and rehabilitation (ISSTD, 2011). The Society has produced separate guidelines for the evaluation and treatment of dissociative symptoms in children and adolescents (Silberg, 2004).

The recommended three-stage approach for treating DID is evidence-based. A longitudinal study that prospectively assesses treatment responses in DID patients in 19 countries has found that over time, patients showed statistically significant reductions in dissociation, PTSD, distress, depression, hospitalisations, suicide attempts, self-harm, dangerous behaviours, drug use and physical pain, as well as higher Global Assessment of Functioning scores (Brand et al., 2013).

DID is often comorbid with anxiety and depression, which can sometimes be lessened with antidepressant medications. These medications have no effect on the DID itself, however (Simon, 1998).

## Other specified dissociative disorder

The catch-all category of ‘**other specified dissociative disorder**’ is important to consider as, in spite of its name, it is likely to be the most common of all the dissociative disorders.

### DSM-5

#### DSM-5 criteria for other specified dissociative disorder (OSDD)

The other specified dissociative disorder category is used in situations in which the presentation does not meet the criteria for any specific dissociative disorder. Examples of presentations that can be specified using the ‘other specified’ designation include the following:

- chronic and recurrent syndromes of mixed dissociative symptoms
- identity disturbance due to prolonged and intense coercive persuasion, such as brainwashing and thought reform
- acute dissociative reactions to stressful events
- dissociative trance, stupor or coma
- Ganser’s syndrome.

Other specified dissociative disorder (OSDD) overlaps significantly with its predecessor in the DSM-IV: ‘dissociative disorders not otherwise specified’ (DDNOS). As OSDD is a new classification there is no information available on its prevalence; however, its frequency is likely to be similar to DDNOS, which has been found to be the most common dissociative disorder in two general population studies, with a prevalence of 4.4 percent in New York State (Johnson et al., 2006) and 8.3 percent in a female-only sample in Turkey (Sar, Akyuz, & Dogan, 2007). It is also the most common dissociative disorder found in clinical settings (Sar, 2011).

#### Chronic and recurrent syndromes of mixed dissociative symptoms

Chronic and recurrent syndromes of mixed dissociative symptoms also include less-than-marked discontinuities in sense of self and alterations of identity, or episodes of possession in an individual who does not report amnesia.

##### Subclinical DID

Chronic and recurrent syndromes of mixed dissociative symptoms includes a subclinical form of dissociative identity disorder (DID), which was known in the DSM-IV as ‘dissociative disorder not otherwise specified — type 1’ (DDNOS-1). In the DSM-5, DDNOS-1 was subsumed into the broader category OSDD. The difference between the DID and subclinical DID (DDNOS-1) is a matter of severity, and together these constitute the chronic complex dissociative disorders (Sar, 2011). Individuals with subclinical DID experience intrusions (emotions, thoughts, actions, speech and impulses) and hear internal dialogue from their personality states, but unlike alters seen in DID, their personality states are not sufficiently distinct to take executive control in daily life without the person’s awareness (Dell, 2009).

The Structured Clinical Interview for Dissociative Disorders (SCID-D) is the gold standard in detecting subclinical DID. The Multidimensional Inventory of Dissociation, a diagnostic tool for all of the dissociative disorders, is the most effective questionnaire for detecting subclinical DID as it contains specific questions about the consciously experienced intrusions from personality states that are characteristic of this type of dissociation (Dell, 2006).

The recommended treatment for subclinical DID is the same three-stage approach outlined for DID (ISSTD, 2011).

#### Identity disturbance due to prolonged and intense coercive persuasion

This form of identity disturbance can occur as a result of prolonged and intense coercive persuasion in individuals who have been subjected to, for example, brainwashing, thought reform, indoctrination while captive, torture, long-term political imprisonment, or recruitment by sects, cults or terrorist organisations. As a result of these experiences, the individuals may present with prolonged changes in, or conscious questioning of, their identity (APA, 2013).

## Acute dissociative reactions to stressful events

Acute dissociative reactions to stressful events include constriction of consciousness; depersonalisation; derealisation; perceptual disturbances, such as time slowing down or objects appearing larger; micro-amnesias; transient stupor; and/or alterations in sensory-motor functioning, such as analgesia or paralysis. Typically symptoms last less than a month and may be as short as a few hours or days (APA, 2013).

## Dissociative trance, stupor or coma

These forms of dissociation occur, when a person loses awareness of immediate surroundings that manifest as profound unresponsiveness or insensitivity to environmental stimuli. This diagnosis is not appropriate if the trance state is a normal part of a broadly accepted collective cultural or religious practice (APA, 2013).

## Ganser's syndrome

Ganser's syndrome is characterised by confusion, auditory and visual hallucinations, amnesia for recent events, sensory and motor conversion, vacant or fixated gaze and giving approximate answers to questions. It is frequently accompanied by dissociative symptoms, such as disorientation, fugue and conversion, and the amnesia experienced may extend to a period of their life as well as for their Ganser-like symptoms. Historically, Ganser's syndrome has been associated with **malinger** (faking or exaggerating a symptom to avoid a responsibility or to achieve some reward). However, growing evidence indicates that in many cases the symptoms are stress induced and unconsciously generated (Drob & Meehan, 2000; Mendis & Hodgson, 2012).

# 5.2 Somatic symptom and related disorders

**LEARNING OUTCOME 5.2** Summarise the symptoms, aetiology and available treatments for somatic symptom and related disorders.

Somatic symptom and related disorders are characterised by physical symptoms that are inconsistent with, or cannot be fully explained by, any underlying general medical or neurologic condition, and are associated with significant distress and impairment including abnormal thoughts, feelings and behaviours in response to these symptoms rather than the absence of a medical explanation for somatic symptoms (APA, 2013). As shown in table 5.2, the DSM-5 includes four major somatic symptom and related disorders: somatic symptom disorder, illness anxiety disorder (previously known as hypochondriasis) conversion disorder and factitious disorder.

**TABLE 5.2** Diagnoses of somatic symptom and related disorders

DSM-5 diagnosis	Description
Somatic symptom disorder	Excessive thought, distress and behaviour related to somatic symptoms
Illness anxiety disorder	Unwarranted fears about a serious illness in the absence of any significant somatic symptoms
Conversion disorder	Neurological symptom(s) that cannot be explained by medical disease or culturally sanctioned behaviour
Factitious disorder	Falsification of psychological or physical symptoms, without evidence of gains from those symptoms

People with somatic symptom and related disorders tend to seek frequent medical treatment, sometimes at great expense. They often see several different physicians for a given health concern and they may try many different medications.

For well over a century, somatoform symptoms and dissociative symptoms have been considered by many as two sides of the same coin with trauma playing an aetiological role in both. As with dissociation, there is a strong, graded relationship between adverse childhood experiences and somatoform and conversion symptoms (Lackner, Gudleski, & Blanchard, 2004; Maaranen, et al., 2004).

### FOCUS ON DISCOVERY 5.3

#### The link between psychological distress during childhood and physical symptoms in adulthood

The idea that psychological distress can manifest in physical symptoms is supported by an increasing body of scientific evidence. The number of different types of adversity a person has experienced during their childhood greatly increases their risk of developing a range of health problems. This occurs as the brain and endocrine system release chemicals during and after stressful events that are useful in ensuring short-term survival, but are harmful to long-term health.

A longitudinal epidemiological study examining childhood adversity — known as Adverse Childhood Experiences (ACE) Study — looks at an individual's exposure to physical, emotional and sexual abuse, physical and emotional neglect and household dysfunction including domestic violence, separation from a parent, incarceration, mental illness and substance abuse. An ACE score is given based on the number of different types of adversity a person has faced during their childhood.

The greater the number of ACEs the greater the risk of physical (and mental) health issues in adulthood, including autoimmune disease, heart disease, diabetes and cancer. The link between childhood adversity and disease remains strong even when controlling for conventional risk factors associated with an unhealthy lifestyle, such as smoking and cholesterol. The impact of adverse experiences on health and wellbeing is so striking that a person with an ACE score of 6 or more has a life expectancy that is nearly 20 years shorter than a person with a score of zero (Larkin, Shields, & Anda, 2012).

Adverse childhood experiences are closely linked to the development of medically unexplained symptoms in adulthood. The risk of experiencing multiple somatic symptoms (defined as six or more symptoms in at least two different organ systems) is increased 2.7-fold if a person has an ACE score of 4 or more (Anda, et al., 2006). ACE scores are predictive of health issues traditionally considered to be somatoform, including chronic fatigue syndrome (Heim et al., 2006), irritable bowel syndrome (Park et al., 2016), headaches (Larkin et al., 2012), fibromyalgia and somatoform pain disorder (Imbierowicz & Egle, 2003).

The ACE studies highlight that trauma and stress play a significant role in the development of a range of illnesses, not only those that are considered to be somatoform.

#### QUESTIONS

1. On average, an individual who has experienced several types of adverse childhood experiences (ACEs) will die 20 years prematurely. What are the reasons for this?
2. If you are a therapist treating somatic symptom and related disorders, what might be the benefits of knowing a patient's ACE score?

A number of factors may contribute to somatic symptom and related disorders, including genetic and biological vulnerability (e.g., increased sensitivity to pain), learning (e.g., attention obtained from illness, lack of reinforcement of non-somatic expressions of distress), and cultural and social norms that devalue and stigmatise psychological suffering as compared with physical suffering. A growing body of research, including the ACE studies outlined in focus on discovery 5.3, shows how common early traumatic experiences, such as violence, abuse and deprivation, are in people with somatoform symptoms (APA, 2013). The quality of the relationship the person had with their primary caregiver in infancy is also predictive of somatisation in adulthood with individuals that have fearful or preoccupied attachment styles reporting significantly more somatic symptoms than individuals with a secure attachment style (Ciechanowski, Walker, Katon, & Russo, 2002).

Somatic symptom disorder and illness anxiety disorder both involve distress and energy expenditure about a health concern. In somatic symptom disorder, the distress revolves around a somatic symptom that exists, whereas in illness anxiety disorder, the distress is about the potential for a medical illness in the absence of significant somatic symptoms. Conversion disorder involves neurological or medical conditions where no organic cause can be found, such as the clinical case presented below in which a woman effectively became blind for over a decade as a result of psychological trauma. This chapter concludes with an overview of factitious disorder, where a person compulsively seeks unnecessary medical attention in a conscious or subconscious attempt to garner care, concern and support from medical staff. In its more extreme form, factitious disorder is often referred to as Munchausen syndrome.

Somatic symptom disorder, illness anxiety disorder and conversion disorder have a significant impact on the person's wellbeing and quality of life. There is a financial and societal cost due to the high cost of health care and the person's reduced capacity to work and to engage meaningfully in family and community life. Research from Australia and New Zealand indicates those who have somatic and/or health anxiety symptoms are more likely to be frequent users of the health care system (Lee, Johnson, Haris, & Sundram, 2016; Pymont & Butterworth, 2015).

Somatic symptoms may begin or intensify after trauma, conflict or stress. To an outside observer, it may seem that the person is using the somatic symptom to avoid some unpleasant activity or to get attention and sympathy. People with somatic symptom disorder frequently have no sense of this as they usually experience their symptoms as completely physical and their distress over their symptoms is authentic.

Concerns about somatic symptoms tend to develop early in adulthood (Cloninger, Martin, Guze, & Clayton, 1986). Although many will experience these concerns throughout their lifetime, the symptoms may wax and wane, and for some, recovery occurs naturally.

Somatic symptom and related disorders tend to co-occur with anxiety disorders, mood disorders, substance use disorders and personality disorders (Kirmayer, Robbins, & Paris, 1994) and in dissociative disorders (Brown et al., 2007; Dell, 2006). For instance, an Australian study found 71 percent of individuals with DID met the diagnostic criteria for somatisation disorder, which is now classified as somatic symptom disorder (Middleton & Butler, 1998). Somatic symptom and related disorders also appear to be more common in women than men (Demyttenaere et al., 2004).

The diagnostic criteria for somatic symptom and related disorders have been criticised for being stigmatising, for the fact that the criteria of health concerns that are a cause of 'excessive anxiety' is subjective, and because these conditions are remarkably varied. For example, some people develop somatic symptoms in the context of anxiety and depressive disorders, whereas others do not (Lieb, Meinschmidt, & Araya, 2007). Some may have medical conditions that provoke the symptoms; others may not.

#### CLINICAL CASE

##### **The blind woman who switched personalities and could suddenly see**

BT had been blind for 17 years. She lost her sight after suffering a traumatic accident as a young woman. Following a series of comprehensive vision tests, doctors had diagnosed her with cortical blindness caused by damage to the visual processing centres in her brain. BT was given a seeing-eye dog to guide her and grew accustomed to her blindness.

BT also had dissociative identity disorder (DID) with 10 vastly different personalities competing for control of her body. It was while seeking treatment for her DID that the ability to see suddenly returned. Not to BT, a 37-year-old German woman, but to a teenage boy she sometimes became. With therapy over the course of months, all but two of BT's identities regained their sight.

When BT alternated between sighted and sightless personalities, her vision switched as well — flicking on and off like a light switch in her mind. The world would appear, then go dark. Malingering (i.e. faking or grossly exaggerating symptoms to avoid a responsibility or achieve some reward) was ruled out by EEG tests. When BT was in her two blind states, her brain showed none of the electrical responses to visual stimuli that sighted people would display — even though BT's eyes were open and she was looking right at them. Two of her identities retain the 'coping mechanism' of blindness in situations that are particularly emotionally intense when she prefers not to be able to see in an attempt to compartmentalise the pain. (Adapted from Kaplan, 2015)



### QUESTIONS

1. Do you think that the therapeutic goal should be to enable BT to have vision in all her alters at all times? Why?
2. What therapeutic approaches do you think would be helpful to BT?

## Neurobiological and cognitive factors that increase awareness of and distress over somatic symptoms

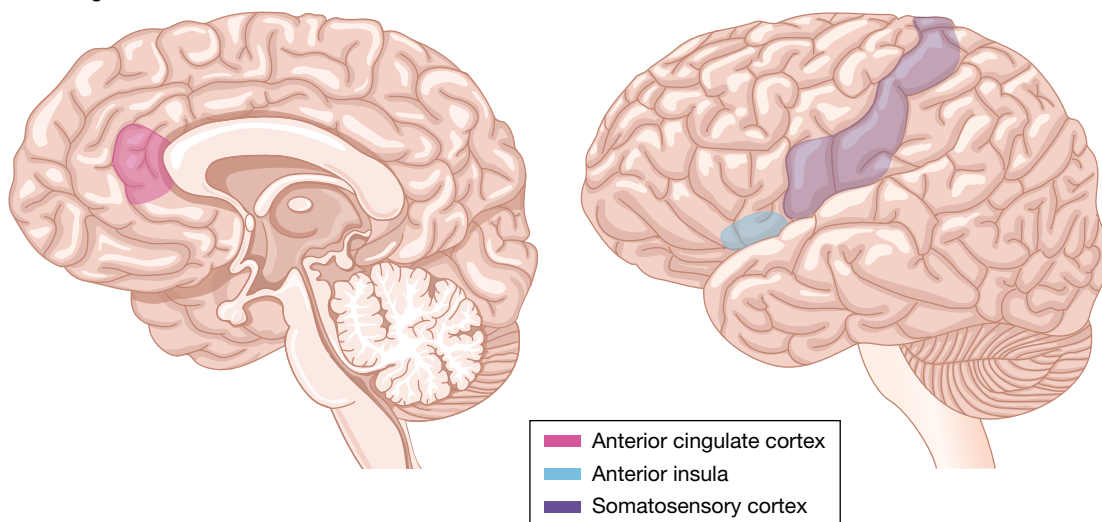
In understanding somatic symptom disorders, then, the key issue is not whether people have some bodily sensation, but rather why some people are more keenly aware of and distressed by these sensations.

Neurobiological models of somatic symptom and related disorders focus on brain regions activated by unpleasant body sensations. Pain and uncomfortable physical sensations, such as heat, increase activity in regions of the brain called the anterior insula and the anterior cingulate cortex (ACC) (Price et al., 2009). These regions have strong connections with the somatosensory cortex, a region of the brain involved with processing bodily sensations (see figure 5.1). Heightened activity in these regions might be related to more intense pain ratings in response to a standardised stimulus (Mayer et al., 2005). Some people, then, may have hyperactive brain regions that are involved in evaluating the unpleasantness of body sensations. This would help explain why they are more vulnerable to experiencing and noticing somatic symptoms and pain.

**FIGURE 5.1** People with somatic symptom and related disorders appear to have increased activity in brain regions implicated in evaluating the unpleasantness of body sensations: the rostral anterior insula, the anterior cingulate and the somatosensory cortex. The anterior cingulate is also involved in depression and anxiety.

a. Midsagittal section

b. Lateral view



In addition to somatic symptoms being related to early adverse experiences, it is well known that pain and somatic symptoms can be increased by anxiety, depression and stress hormones (Gatchel, Peng, Peters, Fuchs, & Turk, 2007). Depression and anxiety are also directly related to activity in the ACC, the area of the brain which is concerned with pain perception and regulation (Wiech & Tracey, 2009), and experiences of emotional pain, such as remembering a relationship breakup, can also activate the ACC and the anterior insula. The involvement of these regions in experiences of physical and emotional

pain may help explain why emotions and depression can intensify experiences of pain (Villemure & Bushnell, 2009).

## Treatment of somatic symptom and related disorders

One of the major obstacles to treatment is that most people with somatic symptom and related disorders usually want medical care, not mental health care. Patients may resent mental health referrals from their physician because they interpret such a referral as a sign that the doctor thinks the illness is ‘all in their head’. Most somatic and pain concerns have both physical and psychological components and so a gentle reminder of the mind–body connection can enhance a patient’s willingness to consider psychological treatment approaches.

Figure 5.2 illustrates the most prominent factors underlying and maintaining somatoform disorders and treatment options. The orange boxes are relevant to understanding how a person might initially develop a somatic symptom. The blue boxes are relevant for understanding reactions to a somatic symptom. Once a somatic symptom develops, two cognitive variables appear important: attention to body sensations and interpretation of those sensations.

Indeed, as shown in previous chapters, physical health concerns are common among people suffering from anxiety or depression, which is also closely linked to trauma, stress and adversity. It should therefore come as no surprise that treating anxiety and depression often reduces somatic symptoms (Phillips, Li, & Zhang, 2002; Smith, 1992). Psychoeducation programs can help patients recognise links between their negative moods and somatic symptoms (Morley, 1997). Techniques such as relaxation training and various forms of cognitive–behavioural treatment have proven useful in reducing depression and anxiety, and these reductions in turn lead to reductions in somatic symptoms (Payne & Blanchard, 1995).

Cognitive–behavioural therapists have applied many different techniques to help people with somatic symptom and related disorders. As outlined in figure 5.2, these techniques include helping people (1) identify and change the emotions that trigger their somatic concerns, (2) change their cognitions regarding their somatic symptoms, and (3) change their behaviours to stop playing the role of a sick person and to gain more reinforcement for engaging in other types of social interactions (Looper & Kirmayer, 2002). More specific information on the treatment of the specific disorders is listed in the relevant section below.

## Somatic symptom disorder

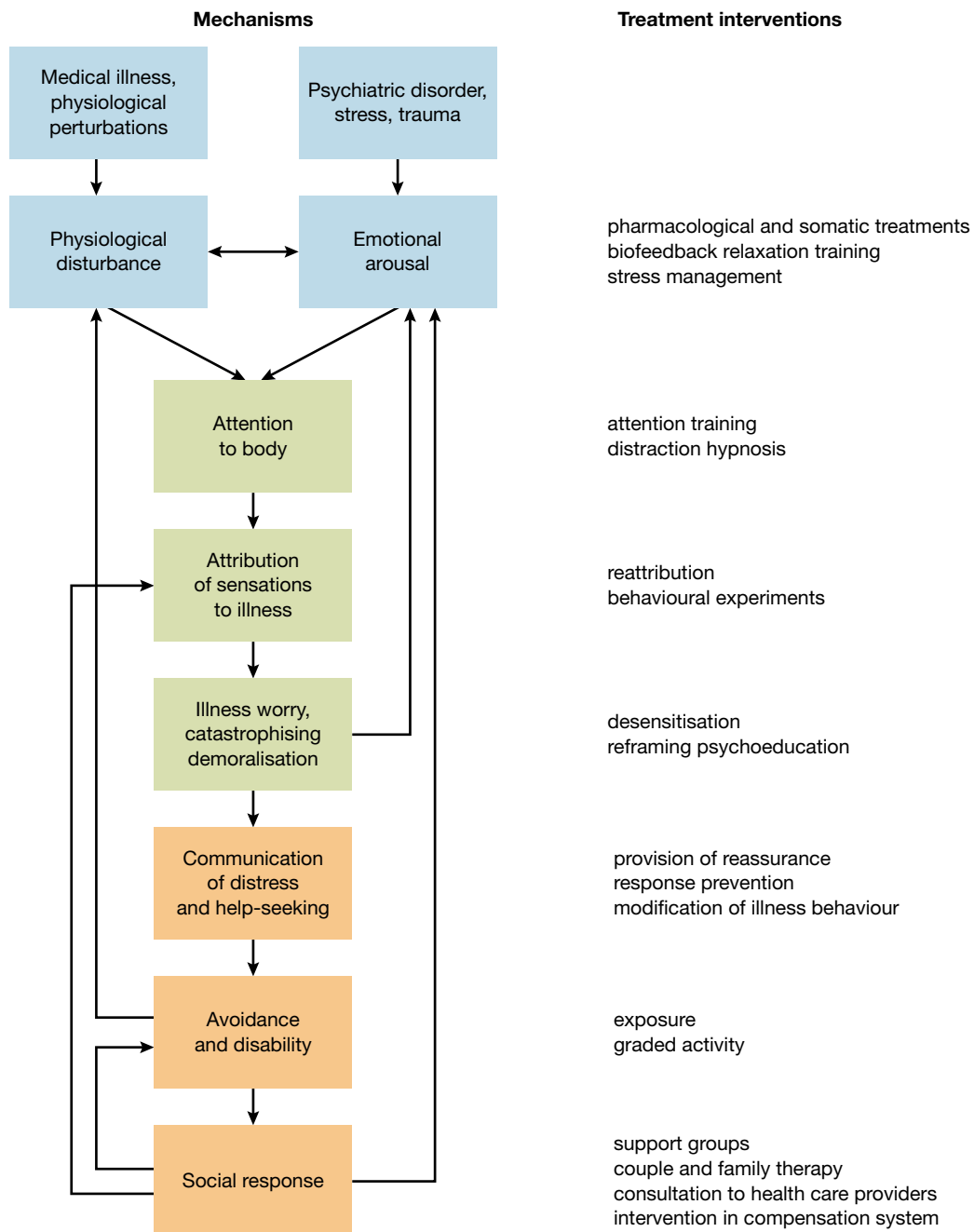
The key feature of **somatic symptom disorder** is excessive anxiety, energy or behaviour focused on somatic symptoms. To be diagnosed, this preoccupation must persist for at least six months. The somatic symptoms can vary substantially. The most frequent somatoform symptoms are back pain, joint pain, pain in extremities and headache, as well as abdominal symptoms, such as bloating or intolerance of several foods and cardiovascular symptoms, such as heart palpitations (Rief, Hessel, & Braehler, 2001). Some might experience a multitude of symptoms from many different body systems while others experience pain as the major concern.

### DSM-5

#### DSM-5 criteria for somatic symptom disorder

- People with somatic symptom disorder experience at least one somatic symptom that is distressing or disrupts daily life.
- People with somatic symptom disorder have excessive thought, distress and behaviour related to somatic symptom(s) or health concerns, as indicated by at least one of the following:
  1. persistently high level of anxiety about health or symptoms
  2. disproportionate and persistent concerns about the seriousness of symptoms
  3. excessive time and energy devoted to these symptoms or health concerns.
- When diagnosing somatic symptom disorder, the person should specify if somatic symptoms:
  1. predominantly involve pain
  2. are severe with marked impairment and have a duration of more than six months.

**FIGURE 5.2** Mechanisms involved in somatic symptom and related disorders



*Source:* Looper and Kirmayer (2002).

The prevalence of somatic symptom disorder in the general population is estimated to be 0.3 percent and is four times more common in females (Rief, Hessel, & Braehler, 2001).

Somatic symptom disorder can be diagnosed regardless of whether symptoms can be explained medically. It is nearly impossible to determine whether some symptoms are biologically caused. Doctors often disagree on whether a symptom has a medical cause (Rief & Broadbent, 2007). Indeed, most

people experience at least one mild physical symptom at some point in their lifetime that cannot be explained medically (Simon, Von Korff, Piccinelli, Fullerton, & Ormel, 1999). Some people might have a condition that defies diagnosis because of limits in medical knowledge and technology. As technology has improved, some conditions that were historically difficult to explain have become better understood. As one example, complex regional pain syndrome was previously believed to be caused by psychological factors, but animal and human research now indicate that these symptoms result from inflammation secondary to autoimmune disorder (Cooper & Clark, 2013). Many common syndromes remain a focus for research because their aetiology is not understood, including irritable bowel syndrome, fibromyalgia, chronic fatigue, non-ulcer dyspepsia and some forms of chronic pain (Cooper & Clark, 2013). The presence of these syndromes is not a reason to diagnose somatic symptom disorders. When psychological factors are the cause of symptoms, an alternative DSM diagnosis, labelled 'psychological factors affecting other medical conditions', may be appropriately considered.

### CLINICAL CASE

#### Maria

Maria, a 32-year-old woman, was referred to a psychologist by her physician. Over a period of about six months, her doctor had seen Maria 23 times for a range of complaints — general aches and pains, bouts of nausea, fatigue, irregular menstruation and dizziness. But various tests, including complete blood workups, x-rays and spinal taps, had not revealed any pathology.

On meeting her therapist, Maria immediately let him know that she was a somewhat reluctant client: 'I'm here only because I trust my doctor and she urged me to come. I'm physically sick and don't see how a psychologist is going to help'. But when Maria was asked to describe the history of her physical problems, she did so without prevarication.

According to Maria, she had always been sick. As a child, she had had episodes of high fever, frequent respiratory infections, convulsions and her first two operations — an appendectomy and a tonsillectomy. During her 20s, Maria had gone from one physician to another. She had suffered with unbearable periods of vomiting. She had seen several gynaecologists for her menstrual irregularity and for pain during intercourse, and she had undergone dilation and curettage (scraping the lining of the uterus). She had been referred to neurologists for her headaches, dizziness and fainting spells and they had performed EEGs, spinal taps and even a CT scan. Other physicians had ordered ECGs for her chest pains. Maria seemed genuinely distressed by her health problems, and doctors responding to her desperate pleas for a cure had performed rectal and gallbladder surgery.

When the interview shifted away from Maria's medical history, it became apparent that she was highly anxious in many situations, particularly those in which she thought she might be evaluated by other people. Indeed, some of her physical symptoms were typical of those experienced by people diagnosed with anxiety disorders.

#### QUESTIONS

1. What are the main factors that would support a diagnosis of somatic symptom disorder in Maria's case?
2. Is it possible that she meets the criteria for a different somatic symptom and related disorder? Why?

A review of the effectiveness of cognitive-behavioural therapy (CBT) for treating somatic illness and related syndromes found it was effective in reducing physical symptoms in 71 percent of studies, yet was only effective in reducing psychological distress in 8 percent of studies. Hence, in the absence of any improvements in psychological distress, somatic symptoms still improved. CBT was generally found to be efficacious with five or more sessions of individual and group therapy. Behavioural therapy, relaxation therapy, biofeedback and antidepressants, even in the absence of depression, have been demonstrated to be beneficial (Kroenke & Swindle, 2000). Symptoms may also improve in response to high levels of warmth, attention and reassurance from doctors (Kaptchuk et al., 2008).

## Somatic symptom disorder with predominant pain (previously known as pain disorder)

In the DSM-IV, diagnosis of pain disorder was given where a person experienced chronic pain that was caused by psychological factors, with or without an associated medical condition. In the DSM-5, pain disorder is no longer a diagnosis in its own right, but is classified as somatic symptom disorder with predominant pain. Chronic pain is very common, with around 37 percent of people in developed countries and 41 percent in developing countries experiencing it during the course of a year. It has been argued that the new approach to pain disorder in the DSM-5 is overly inclusive and many medical patients with significant symptoms of emotional distress will receive an inappropriate psychiatric diagnosis (Katz, Rosenbloom, & Fashler, 2015).

Research supports the use of CBT for somatic symptom disorder with pain (Ehde, Dillworth, & Turner, 2014) and has been shown to reduce activity in regions of the brain that are involved in responding to pain stimuli (Flor, 2014). A variant of CBT known as acceptance and commitment therapy (ACT) that encourages the client to view pain, suffering and moments of depression and anxiety as a natural part of life (McCracken & Vowles, 2014) has been found to be as helpful as standard CBT (Veehof, Oskam, Schreurs, & Bohlmeijer, 2011). Individuals with chronic pain can be trained to use cognitive strategies to alter pain perception while viewing pain-related brain activity on an fMRI. Voluntary control over activation in this region was powerful enough to impact severe, chronic clinical pain (deCharms et al., 2005). Hypnosis appears to help reduce pain levels and has been shown to influence brain regions involved in experiencing and interpreting pain (Jensen & Patterson, 2014).

Antidepressants can be helpful in relieving pain and associated distress (Fishbain, Cutler, Rosomoff, & Rosomoff, 2000), even when given in dosages too low to alleviate the associated depression (Simon, 1998). Antidepressants are preferred over opioid medications, which are highly addictive (Streltzer & Johansen, 2006).

## Illness anxiety disorder

The main feature of **illness anxiety disorder**, formerly called *hypochondriasis*, is a preoccupation with fears of having a serious disease despite having no significant symptoms. To meet the DSM criteria for diagnosis, these fears must lead to excessive care seeking or maladaptive avoidance behaviours that persist for at least six months. People with this disorder are easily alarmed about their health and they tend to worry about the possibility of cancer, heart attacks, AIDS and strokes (Rachman, 2012) and may be haunted by powerful visual images of becoming ill or dying (Muse, McManus, Hackmann, & Williams, 2010). They may react with anxiety when they hear about illnesses in their friends or in the broader community. These fears are not easily calmed and others may become frustrated when attempts to soothe worries fail.

### DSM-5

#### DSM-5 criteria for illness anxiety disorder

- Preoccupation with and high level of anxiety about having or acquiring a serious disease.
- Excessive illness behaviour (e.g., checking for signs of illness, seeking reassurance) or maladaptive avoidance (e.g., avoiding medical care).
- No more than mild somatic symptoms are present.
- Not explained by other psychological disorders.
- Preoccupation lasts at least six months.

The prevalence of illness anxiety disorder has been found to be fairly low (between 0.05 percent and 0.4 percent) in general population studies, although subclinical levels of the disorder are not uncommon (between 2 percent and 6 percent). Severe anxiety over health concerns persists for at least five years in over two-thirds of those with the disorder (Barsky, Fama, Bailey, & Ahern, 1998).

People prone to worries about their health also tend to interpret their physical symptoms in the worst possible way (Rief & Broadbent, 2007). A small physical sign is interpreted as a sign of impending catastrophe. For example, interpreting a red blotch on the skin as a sign of cancer (Marcus, Gurley, Marchi, & Bauer, 2007) or overestimating the odds that a symptom is a sign of a disease (Rief, Buhlmann, Wilhelm, Borkenhagen, & Brähler, 2006). The exact form of the cognitive bias may vary, but once these negative thoughts begin, the resultant elevations of anxiety and cortisol may exacerbate somatic symptoms and distress over those symptoms (Rief & Auer, 2001). Focusing attention on the body may also increase awareness of unusual physical sensations that would otherwise have gone unnoticed.

In a previous chapter, we described a very similar cognitive process as part of panic disorder. That is, people with panic disorder are likely to overreact to physiological symptoms. In panic disorder, the person often believes that the symptoms are a sign of an immediate threat (e.g., a heart attack), whereas in somatic symptom disorder, the person believes the symptoms are a sign of an underlying long-term disease. Illness anxiety disorder and obsessive compulsive disorder (OCD) also share clinical similarities of fears that are often recurrent and intrusive and behaviours that are carried out to reduce distress, such as self-checking and reassurance seeking (Harding, Skritskaya, Doherty, & Fallon, 2008). Not surprisingly, illness anxiety disorder often co-occurs with anxiety and mood disorders (Noyes, 1999). In fact, 45 percent of patients with illness anxiety disorder also met the criteria for an anxiety or depressive disorder (Simon, Gureje, & Fullerton, 2001).

Somatic symptom disorder and illness anxiety disorder both involve health anxiety. When the fears about a serious disease are accompanied by somatic symptoms, the appropriate DSM-5 diagnosis is somatic symptom disorder. Because so few people with intense fears about their health are free of somatic symptoms, very few people are expected to meet the criteria for illness anxiety disorder. Somatic symptom disorder is expected to be three times as common as illness anxiety disorder (Heller et al., 2013).

Although people with these disorders are the most frequent consumers of medical care, they are often dissatisfied when no medical explanation or cure can be identified. They may view their physicians as incompetent and uncaring (Persing, Stuart, Noyes, & Happel, 2000). Despite their negative assessment of the medical profession, they will often continue extensive treatment seeking, visiting new doctors and requesting new tests. Many patients become unable to work because of the severity of their concerns. One way of identifying illness anxiety disorder is to inform the treating general practitioner (GP) when a patient appears to be an intensive user of health care services so that they can minimise the use of diagnostic tests and medications. These types of interventions with physicians can reduce the use of costly health care services (Konnopka et al., 2012).

The tendency to be overly concerned about one's health may have evolved from early experiences of medical symptoms or from family attitudes to physical illness. Consistent with these childhood influences on cognitive biases, people with somatic symptom and related disorders report that as children they often missed school because of illness (Barsky, Brener, Coeytaux, & Cleary, 1995).

Fear that a bodily sensation signifies illness is likely to have two behavioural consequences. First, the person may assume the role of being sick and avoid work, exercise and social tasks (Martin & Jacobi, 2006) and these avoidant behaviours in turn can intensify symptoms by limiting other healthy behaviours. Second,



the person may seek reassurance from doctors and from family members and this help-seeking behaviour may be reinforced if it results in the person getting attention or sympathy. Often, people with these disorders experience depression and interpersonal insecurity, so they may find attention and sympathy for health concerns particularly reinforcing (Rief & Broadbent, 2007). People may receive other types of behavioural reinforcers for somatic symptoms, such as disability payments. Hence, many different behavioural factors can maintain and intensify health anxiety.

The two most effective treatment modalities for hypochondriasis are CBT and selective serotonin reuptake inhibitors (SSRIs) (Greeven et al., 2007; Harding et al., 2008). CBT is proven to be effective in reducing health concerns, depression, anxiety and health care utilisation (Thomson & Page, 2007). Behavioural techniques might help people resume healthy activities and rebuild a lifestyle that has been damaged by too much focus on illness-related concerns (Warwick & Salkovskis, 2001). Mindfulness meditation is also promising, with participants learning this technique reporting less health anxiety than those with standard medical care, and these improvements being sustained one year later (McManus, Surawy, Muse, Vazquez-Montes, & Williams, 2012).

### CLINICAL CASE

#### Louis

Louis, a 66-year-old man, was referred to a psychiatrist by his cardiologist because of health anxiety. Louis was depressed and anxious for years, but was far more concerned about his potential for heart problems. Several years before, he had developed intermittent symptoms of heart palpitations and chest pressure. Extensive medical tests were within the normal range, yet he continued to seek additional tests and to monitor the results carefully. He gathered a thick file of articles on cardiovascular conditions, adopted strenuous diet and exercise routines, stopped all activities that might be challenging to his heart, such as travel and sex, and retired early from running his restaurant. By the time he sought treatment, he was measuring his blood pressure four times a day using two machines so that he could average the readings, and was keeping extensive logs of his blood pressure readings.

Before treatment could begin, Louis had to understand that the way he was thinking about his physical symptoms was intensifying those physical symptoms as well as creating emotional distress. His therapist taught him a model of symptom amplification, in which initial physical symptoms are exacerbated by negative thoughts and emotions. The therapist used statements such as 'A headache you believe is due to a brain tumour hurts much more than a headache you believe is due to eye strain'. Once Louis understood that his thoughts and behaviour might be increasing his medical concerns, treatment focused on four goals. First, Louis was coached to identify one doctor with whom to routinely discuss health concerns and to stop seeking multiple medical opinions. Second, Louis was taught to reduce the time spent engaging in excessive illness-related behaviours, such as logging his blood pressure. His therapist showed him that these behaviours were actually increasing his anxiety rather than providing relief. Third, Louis was taught to consider the thoughts he had in response to his symptoms, which tended to be very negative and pessimistic. For example, the therapist and Louis identified ways in which he tended to catastrophise harmless physical sensations by viewing them as evidence for

People with health anxiety are not easily reassured that they are well, even when extensive medical tests indicate no problems.



heart disease. Louis was taught to consider more benign reasons for his physical symptoms. Finally, Louis was encouraged to build other aspects of his life in order to diminish the focus on physical symptoms and in response, he began to consult for restaurants. Taken together, these interventions helped Louis reduce his anxiety, diminish his focus on and concern about his health, and begin to lead a more enjoyable life. (Adapted from Barsky, 2006)

### QUESTIONS

1. What role could Louis' GP have played in the identification and treatment of his illness anxiety disorder?
2. What other approaches could his therapist have used to reduce his symptoms?

## Conversion disorder

**Conversion disorder**, also called functional neurological symptom disorder, is a condition in which psychological stress following a traumatic or stressful experience converts to a physical impairment. Conversion symptoms manifest in a variety of ways, but do not have a neurological or organic basis. Motor symptoms include weakness or paralysis, and abnormal movements, such as tremors, involuntary movements, repetitive movements, abnormal postures and gait abnormalities. Sensory symptoms include altered, reduced or absent skin sensation, vision or hearing. Episodes of abnormal generalised limb shaking with apparent impairment or loss of consciousness may resemble epileptic seizures (these are known as pseudoseizures or psychogenic non-epileptic seizures). There may be episodes of unresponsiveness resembling fainting or coma. Other symptoms include reduced or absent speech volume (dysphonia/aphonia), altered articulation (dysarthria) or a sensation of a lump in the throat (globus) (APA, 2013). Many people with conversion disorder do not connect their symptoms with their stressful situations.

Three factors are thought to contribute to or cause conversion disorder — psychodynamic factors, learning theory and biological factors. This disorder is described in the earliest writings on psychological disorders. *Hysteria* was the term originally used to describe the disorder, which the Greek physician Hippocrates considered to be an affliction limited to women and brought on by the wandering of the uterus through the body. (The Greek word *hystera* means 'womb'; the wandering uterus symbolised the longing of the woman's body for the production of a child.) The term *conversion* originated with Sigmund Freud, who thought that anxiety and psychological conflict were converted into physical symptoms. Freud's psychodynamic theory paved the way for the idea of conversion symptoms as it is based on the premise that symptoms manifest as a result of unconscious conflict between a forbidden wish of the patient and his or her conscience. The conversion symptoms symbolically represent a partial wish fulfilment without the individual's full awareness of the unacceptable desire. For example, paralysis of the limbs may prevent the person expressing repressed anger. Learning theory is based on the premise that symptoms of illness learned in childhood are called forth as a means of coping with an impossible situation. Biological factors are apparent where symptoms are caused by impaired cerebral hemispheric communications and excessive cortisol arousal that inhibit the individual's awareness of bodily sensations. The patient may also exhibit subtle impairments on neuropsychological tests (Ali et al., 2015).

### DSM-5

#### DSM-5 criteria for conversion disorder

- People with conversion disorder exhibit one or more symptoms affecting voluntary motor or sensory function.
- Clinical findings provide evidence of incompatibility between the symptom and recognised neurological or medical conditions.
- Symptoms cause significant distress or functional impairment or warrant medical evaluation.
- Specify if the symptom includes: weakness or paralysis; abnormal movement; swallowing symptoms; speech symptom; attacks or seizures; anaesthesia or sensory loss; sensory symptom (e.g., visual, olfactory or hearing disturbance); or mixed symptoms.

The prevalence of conversion disorder in the general population is less than 1 percent (Faravelli, Salvatori, Galassi, & Aiazzi, 1997), which rises to 5 percent in general hospital settings (Ali et al., 2015) and to 8 percent in patients visiting neurology clinics (Hubschmid, et al., 2015). Conversion disorder is between two and ten times more frequent in women and is far more common in young people. It is also more common in people with low socioeconomic status, living in a rural area, having a low education level and living in a developing country or region (Ali et al., 2015).

Symptoms of conversion disorder usually develop in adolescence or early adulthood, typically after a major life stressor. An episode may end abruptly, but sooner or later the disorder is likely to return, either in its original form or with a different symptom. The prognosis for those with motor impairment or seizures is poor with only 50 percent improving and one-third needing to retire from the workforce early (Hubschmid, et al., 2015).

Patients with conversion disorder are highly likely to meet criteria for another somatic symptom disorder (Brown et al., 2007) and about half meet criteria for a dissociative disorder (Sar, Akyuz, Kundakci, Kiziltan, & Dogan, 2004). Other common comorbid disorders include major depressive disorder, substance use disorders and personality disorders (Brown et al., 2007), mood disorders, generalised anxiety disorder, phobia, obsessive-compulsive disorder, post-traumatic stress disorder and schizophrenia (Ali et al., 2015). Factitious disorder or malingering are distinct from conversion disorder (Allin, Streeruwitz, & Curtis, 2005). To distinguish between genuine and falsified symptoms, clinicians must determine whether the symptoms have been consciously or unconsciously adopted. In malingering, the symptoms are under voluntary control. In conversion disorder, despite the lack of a definitive organic diagnosis, the individual's distress is very real and the physical symptoms the patient is experiencing cannot be controlled at will (Ali et al., 2015).

When a patient reports a neurological symptom, it is important to assess whether that symptom has a true neurological basis. Although some diagnostic distinctions are easy, the clinician still has to be careful in making this diagnosis. It is estimated that genuinely physical problems are misdiagnosed as conversion disorder about 4 percent of the time (Stone et al., 2005). Sometimes behavioural tests can help make this distinction. For example, arm tremors might disappear when the person is asked to move the arm rhythmically. Leg weakness might not be consistent when tested with resistance (Stone, LaFrance, Levenson, & Sharpe, 2010). In one form of conversion disorder, people report tunnel vision, which is incompatible with the biology of the visual system. In another example, people might show a seizure-like event at the same time that a normal EEG pattern is recorded (Stone et al., 2010).

Some symptoms that might seem medically implausible have been shown to have a biological basis. Consider, for instance, the classic example of 'glove anaesthesia', in which the person experiences little or no sensation in the part of the hand and lower arm that would be covered by a glove. For years this was considered a textbook illustration of anatomical nonsense because the nerves run continuously from the hand up the arm. Yet now it appears that carpal tunnel syndrome, a recognised medical condition, can produce symptoms similar to those of glove anaesthesia. Nerves in the wrist run through a tunnel formed by the wrist bones and membranes. The tunnel can become swollen and may pinch the nerves, leading to tingling, numbness and pain in the hand. People who use computer keyboards for many hours a day seem to be at risk for this condition. Beyond glove anaesthesia, other symptoms that would intuitively seem difficult to explain medically, such as the perception of a burning sensation when touching a cold object, have clear medical explanations (in this case, ciguatera, a disease caused by eating certain reef fish). To enhance the reliability of conversion disorder, the DSM-5 provides guidance to clinicians about how to assess whether symptoms might be medically unexplained.

Treatment of conversion disorder emphasises physical therapy combined with behavioural reinforcement strategies, stress management or problem solving (Looper & Kirmayer, 2002). Individual and group psychotherapy and behavioural therapy are widely recommended for treating conversion symptoms. CBT informed psychotherapy and early intervention involving joint psychiatric and neurological consultation have been shown to be effective in reducing physical and psychological symptoms and hospital stays for those with motor symptoms or pseudoseizures (Hubschmid, et al., 2015). Hypnosis,

biofeedback and relaxation may also be of benefit, although more research is needed. Antidepressants or anti-anxiety medication may assist in reducing psychological symptoms, and physical therapies, such as physiotherapy, can improve physical symptoms (Ali et al., 2015).

#### CLINICAL CASE

##### Cynthia's unexplained seizures

Cynthia, a 48-year-old woman, had been having seizures since she was 39. In childhood Cynthia had suffered extensive, sustained and at times bizarre childhood sexual and physical abuse at the hands of her mother and stepfather, which was corroborated by her sister. Cynthia's seizures would be preceded by a headache lasting several hours, followed by palpitations and a sensation of anxiety and then she would lose awareness of herself and her surroundings. Magnetic resonance imaging (MRI) of the brain and an electroencephalogram (EEG) were normal and anti-epileptic medication did not decrease the frequency of her seizures. Cynthia also had multiple psychiatric admissions for depression, agitation and paranoia. Her frequent seizures were also monitored in the psychiatric hospital and, as no epileptiform changes on the EEG were found, she was diagnosed with pseudoseizures. Cynthia was also diagnosed with a dissociative disorder with fugue states and amnesias. In recognition of the psychological basis of her symptoms, Cynthia's prescribed treatment was to continue with individual and group therapy and discontinue anti-epileptic medication. (Adapted from Harden, 1997)

#### QUESTIONS

1. What three factors clarified that there was no neurological or organic cause for the seizures?
2. Explain why Cynthia's abusive childhood is relevant to the diagnosis of conversion disorder.

## Factitious disorder

In **factitious disorder**, people intentionally produce physical or psychological symptoms to assume the role of a patient. Factitious disorder behaviours include providing a false medical history; simulating physical symptoms; modifying physiology to create physical signs; and inducing physical illness. Some will take extraordinary measures to make themselves ill, including injuring themselves, taking damaging medications or injecting themselves with toxins. More severe and chronic forms of factitious disorder are known as Munchausen syndrome, although the terms are often used interchangeably.

Factitious disorder, where the sole goal often seems to be to adopt the patient role, is distinct from malingering in which a person intentionally fakes or greatly exaggerates a symptom to avoid a responsibility, such as work or military duty, or to achieve some reward, such as being awarded an insurance settlement.

Among patients in hospital settings, it is estimated that about 1 percent of individuals have presentations that meet the criteria for factitious disorder (APA, 2013).

#### DSM-5

##### DSM-5 criteria for factitious disorder

- Individuals with factitious disorder falsify physical or psychological signs or symptoms, or induction of injury or disease, associated with identified deception.
- The individual presents himself or herself to others as ill, impaired or injured.
- The deceptive behaviour is evident even in the absence of obvious external rewards.
- In 'factitious disorder imposed on another', the person fabricates or induces symptoms in another person and then presents that person to others as ill, impaired or injured.

What causes factitious disorder? In an overwhelming number of Munchausen case studies, some traumatic event occurred to the person during childhood, including physical or sexual assault and incest, witnessing a relative or close friend be killed, parental divorce and chronic illness, hospitalisation or institutionalisation. In the majority of cases the patient's family responded with ambivalence or over-protection instead of providing adequate social support or reinforcing effective coping strategies. It is theorised that a visit to the doctor or an admission to a hospital may have exposed the child to a more stress-free environment as well as care, concern and support from medical staff. The child may learn that their own parents are more responsive to health complaints and this allows the child to gain access to a hospital and stop the abuse at home. Those with Munchausen syndrome often idealise medical professionals and it is thought they may be subconsciously projecting their wish for a kind and competent parental figure onto doctors and other medical staff. The pattern of dealing with stressful situations by seeking hospitalisation then manifests as a factitious disorder in adulthood (Trask & Sigmon, 1997).

Although there are limited psychological and psychiatric evaluations of Munchausen patients, case studies indicate poor ego functioning, including a fear of abandonment, the use of primitive idealisation and self-destructive behaviour. Feelings of abandonment are often coupled with thoughts of destructive rage, fears of retaliation and inappropriate attachment to others in the person's environment (Mayo & Haggerty, 1984).

#### RESEARCH EXAMPLE

##### Miss Scott

In one of the most severe examples of factitious disorder that has been reported, a woman named Miss Scott described being hospitalised at more than 600 hospitals and having 42 operations, nearly all of which were not needed (Grady, 1999). Some days she would leave one hospital and be admitted to a different hospital by nightfall. One doctor who examined the scars on her abdomen reported that 'she looked as if she had lost a duel with Zorro'. When asked about her treatment seeking, she reported, 'To begin with, it was just something I did when I needed someone to care about me. Then it became something I had to do. It was as if something took me over. I just had to be in the hospital. I had to'. She had grown up as an abused, lonely child and one of her early positive memories was the care she received from a nurse after having her appendix removed. After that experience, she once walked into her local hospital feigning a stomach ache, hoping that someone would care about her experience. She spent several days there appreciating the attention that she received. Over the course of the next year, she began to seek care at a series of different hospitals. 'Soon she was spending all her time hitchhiking from town to town, trying to get into the hospital' (Grady, 1999, p. D5). For Miss Scott, being a patient became her chief way of gaining support and nurturance.

##### QUESTIONS

1. In what ways was Miss Scott's childhood similar to others diagnosed with factitious disorder?
2. What factors in Miss Scott's case enable you to distinguish factitious disorder from malingering?

#### Factitious disorder imposed on another

The diagnosis of 'factitious disorder imposed on another', which is also known as Munchausen-by-proxy, is given when an individual falsifies illness in another, including children, an adult or a pet. The perpetrator, not the victim, is given the diagnosis. The actions of the perpetrator may constitute criminal behaviour and if the victim is a child, it is a form of child abuse and maltreatment.

Therapy for patients with factitious disorder is overwhelmingly ineffective and no evidence-based treatments have been found (Baig, Levin, Lichenthal, Boland, & Breitbart, 2016; Trask & Sigmon, 1997).

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## SUMMARY

### **5.1 Summarise the symptoms, aetiological models and available treatments for dissociative disorders.**

Dissociative disorders are characterised by the disruption and discontinuity in the normal integration of consciousness, memory, identity, emotion, perception, body representation, motor control and behaviour. In depersonalisation/derealisation disorder, the person's perception of the self and surroundings is altered and they may feel detached from their body or may perceive the world as being unreal. Dissociative amnesia is defined by inability to recall important personal experiences of a traumatic or highly stressful nature. The person with dissociative identity disorder (DID) has two or more distinct personalities, each with unique memories, behaviour patterns and relationships, whereas in subclinical DID the personality states intrude on the person's thoughts, feelings and actions, but are not developed enough to take full control of the individual.

Dissociative disorders are related to childhood abuse, neglect, insecure attachment to the primary caregiver, and exposure to traumatic and stressful events. The aetiology of dissociative disorders continues to be debated, though over time, given the increased data available on familial and institutional child abuse, allied with intriguing neuro-physiological findings, most experts see trauma as a major factor. The trauma model suggests that dissociation becomes a reflex coping strategy when the options of fighting or fleeing are not possible, as is frequently the case in young children, living with inescapable repetitive trauma. The fantasy model suggests that fantasy-prone and suggestible individuals, often with genuine histories of abuse, have poor sleep and this combination of factors results in two false beliefs — first, in 'recovered' memories of (more) childhood abuse, and second, that they have a dissociative disorder, when in reality the symptoms and stories of dissociation and trauma are borrowed from the media or other people. The fantasy model has been demonstrated to lack robust empirical support. It is not uncommon for people with histories of a trauma to experience an extended period of time in which they cannot recall it; that all types of memory are open to distortion and recovered memories of trauma are found to be as accurate as continuous memories of trauma. Furthermore, differences in brain activity can be witnessed between alters, yet no differences are seen in people role-playing DID.

In the safe, supportive context of therapy, patients are encouraged to learn new strategies for coping with emotions, so as to gain better control over tendencies to rely on dissociation.

### **5.2 Summarise the symptoms, aetiology and available treatments for somatic symptom and related disorders.**

The common feature of somatic symptom and related disorders is the excessive focus on physical symptoms. The nature of the concern, however, varies by disorder. Somatic symptom disorder is defined by excessive thought, distress and behaviour related to somatic symptoms or health concerns. Illness anxiety disorder is defined by fears of a serious disease when no more than mild somatic symptoms are present. Conversion disorder is characterised by sensory and motor dysfunctions that are incompatible with recognised neurological or medical conditions. Somatic symptom and related disorders may arise suddenly in stressful situations. Factitious disorder can be distinguished from the other somatic symptom disorders as the person compulsively seeks unnecessary medical attention, not because they believe they are unwell, but in a conscious or subconscious attempt to garner care, concern and support from medical staff.

A growing body of research confirms that adverse experiences in childhood are closely linked to a range of illnesses and medical conditions, including those associated with somatic symptom and conversion disorder. Neurobiological models suggest that some people may have a propensity towards hyperactivity in those regions of the brain involved in evaluating the unpleasantness of somatic sensations, including the anterior cingulate and the anterior insula. Cognitive-behavioural models focus on attention to and interpretation of somatic symptoms as a way of understanding why some people

experience such intense anxiety about their health. Psychodynamic theories of conversion disorder have focused on the idea that psychological stress following a traumatic or stressful experience converts to a physical impairment, yet most people are unaware of the subconscious motivation for their symptoms.

Cognitive-behavioural therapy is the most evidenced-based approach for reducing the physical symptoms of somatic symptom and conversion disorder, as well as reducing anxiety in people with illness anxiety disorder.

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## KEY TERMS

**conversion disorder** a disorder in which psychological stress following a traumatic or stressful experience converts to a sensory or motor function impairment, even though there is no detectable neurological or organic explanation for the deficits

**depersonalisation disorder** a dissociative disorder in which the individual has disconcerting and disruptive experiences of being detachment from their mental processes or body, as though observing themselves from outside their body

**derealisation disorder** a dissociative disorder in which the individual has disconcerting and disruptive experiences of their surroundings not being real

**dissociative amnesia** a dissociative disorder in which the person experiences an inability to remember important autobiographical information, usually of a traumatic or stressful nature, to an extent that cannot be explained by ordinary forgetfulness

**dissociative disorders** disorders in which the normal integration of consciousness, memory, or identity is suddenly and temporarily altered; include dissociative amnesia, dissociative identity disorder (formerly known as multiple personality disorder), depersonalisation/derealisation disorder, and other specified dissociative disorder

**dissociative identity disorder (DID)** a dissociative disorder (formerly called multiple personality disorder, or MPD) in which two or more distinct and separate personalities are present within the same individual, each with his or her own memories, relationships and behaviour patterns

**factitious disorder** a disorder in which the individual's physical or psychological symptoms appear under voluntary control and are adopted merely to assume the role of a sick person; called factitious disorder by proxy or Munchausen syndrome by proxy when a parent produces a physical illness in a child

**fantasy model** a model that conceptualises dissociative disorders as primarily the result of fantasy proneness, suggestibility and cognitive failures

**fugue** a subtype of dissociative amnesia disorder in which the person experiences total amnesia, moves, and establishes a new identity

**iatrogenic** caused by medical examination or treatment

**illness anxiety disorder** a disorder defined by a preoccupation with, and high level of anxiety about, having or acquiring a serious disease despite symptoms being mild or non-existent

**malinger** intentionally faking or exaggerating a symptom to avoid a responsibility, such as work or military duty, or to achieve some reward, such as being awarded an insurance settlement

**other specified dissociative disorder** a dissociative disorder diagnosis where the presentation does not meet the criteria for any specific dissociative disorder

**somatic symptom disorder** a disorder defined by excessive thought, distress and behaviour related to at least one somatic symptom that is distressing or disrupts daily life

**trauma model** a model that conceptualises dissociative disorders as a reaction to traumatic stress or severe psychological adversity

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## WEBSITES

1. The International Society for the Study of Trauma and Dissociation (ISSTD) provides a range of resources about the prevalence, consequences of, and treatment for chronic trauma and dissociation. It also provides expert guidelines on treating dissociative identity disorder in adults and in children. ([www.isst-d.org](http://www.isst-d.org))
2. Adverse Childhood Experiences Study (ACEs), Centers for Disease Control and Prevention provides the Adverse Childhood Experiences questions so readers can calculate an ACE score and examine the implications for health and resilience. ([www.cdc.gov/violenceprevention/acestudy/about.html](http://www.cdc.gov/violenceprevention/acestudy/about.html))

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## REFERENCES

- Aderibigbe, Y. A., Bloch, R. M., & Walker, W. R. (2001). Prevalence of depersonalization and derealization experiences in a rural population. *Social Psychiatry and Psychiatric Epidemiology*, 36(2), 63–69.
- Ali, S., Jabeen, S., Pate, R. J., Shahid, M., Chinala, S., Nathani, M., & Shah, R. (2015). Conversion disorder — mind versus body: a review. *Innovations in Clinical Neuroscience*, 12(5–6), 27–33.
- Allen, J. G. (2001). *Traumatic relationships and serious mental disorders*. New York: John Wiley & Sons.
- Allin, M., Streeruwitz, A., & Curtis, V. (2005). Progress in understanding conversion disorder. *Neuropsychiatric Disease and Treatment*, 1(3), 205–209.
- American Psychiatric Association. (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.) Washington DC: American Psychiatric Publishing.
- Anda, R. F., Felitti, V. J., Bremner, J. D., Walker, J. D., Whitfield, C., Perry, B. D., . . . Giles, W. H. (2006). The enduring effects of abuse and related adverse experiences in childhood. A convergence of evidence from neurobiology and epidemiology. [Research Support, Non-US Gov't Research Support, US Gov't, P H S]. *European Archives of Psychiatry and Clinical Neuroscience*, 256(3), 174–186.
- Andreano, J. M., & Cahill, L. (2006). Glucocorticoid release and memory consolidation men and women. *Psychological Science*, 17, 466–470.
- Baig, M. R., Levin, T. T., Lichtenthal, W. G., Boland, P. J., & Breitbart, W. S. (2016). Factitious disorder (Munchausen's syndrome) in oncology: case report and literature review. *Psycho-Oncology*, 25(6), 707–711. doi: 10.1002/pon.3906
- Barsky, A. J., Brener, J., Coeytaux, R. R., & Cleary, P. D. (1995). Accurate awareness of heartbeat in hypochondriacal and non-hypochondriacal patients. *Journal of Psychosomatic Research*, 39, 489–497.
- Barsky, A. J., Fama, J. M., Bailey, E. D., & Ahern, D. K. (1998). A prospective 4- to 5-year study of DSM-III-R hypochondriasis. *Archives of General Psychiatry*, 55, 737–744.
- Barsky, A. (2006). "Doctor, are you sure my heart is okay?" Cognitive-behavioral treatment of hypochondriasis. In R. L. Spitzer, M. B. W. First, J. B. Williams & M. Gibbon (Eds.), *DSM-IV-TR® casebook, volume 2: Experts tell how they treated their own patients* (pp. 251–261). Washington, DC: American Psychiatric Association.
- Bernstein-Carlson, E. B., & Putnam, F. W. (1993). An update on the dissociative experiences scale. *Dissociation*, 6.
- Borch-Jacobsen, M. (1997, April 24). Sybil: The making of a disease? An interview with Dr. Herbert Spiegel. *New York Review of Books*, 44(7), 60.
- Brand, B. L., McNary, S. W., Myrick, A. C., Classen, C., Lanius, R., Loewenstein, R. J., et al. (2013). Supplemental material for a longitudinal naturalistic study of patients with dissociative disorders treated by community clinicians. *Psychological Trauma: Theory, Research, Practice, and Policy*. Advance online publication.
- Brand, B. L., Myrick, A. C., Loewenstein, R. J., Classen, C. C., Lanius, R., McNary, S. W., et al. (2012). A survey of practices and recommended treatment interventions among expert therapists treating patients with dissociative identity disorder and dissociative disorder not otherwise specified. *Psychological Trauma: Theory, Research, Practice, and Policy*, 4, 490–500.
- Brand, B. L., Sar, V., Stavropoulos, P., Krüger, C., Korzekwa, M., Martínez-Taboas, A., & Middleton, W. (2016). Separating fact from fiction: An empirical examination of six myths about dissociative identity disorder. *Harvard Review of Psychiatry*, 24(4), 257–270. doi: 10.1097/hrp.0000000000000100
- Brewin, C. R., Ma, B. Y., & Colson, J. (2013). Effects of experimentally induced dissociation on attention and memory. *Consciousness and Cognition*, 22(1), 315–323.
- Briere, J., & Conte, J. (1993). Self-reported amnesia for abuse in adults molested as children. *Journal of Traumatic Stress*, 6(1), 21–31. doi: 10.1002/jts.2490060104
- Brown, A. S., Bottiglieri, T., Schaefer, C. A., Quesenberry, C. P., Jr., Liu, L., Bresnahan, M., & Susser, E. S. (2007). Elevated prenatal homocysteine levels as a risk factor for schizophrenia. *Archives of General Psychiatry*, 64, 31–39.
- Brown, R. J., Cardena, E., Nijenhuis, E., Sar, V., & Van der Hart, O. (2007). Should conversion disorder be reclassified as dissociative disorder in DSM-5? *Psychosomatics*, 48, 369–378.

- Carlson, V., Cicchetti, D., Barnett, D., & Braunwald, K. (1989). Disorganized/disoriented attachment relationships in maltreated infants. *Developmental Psychology*, 25(4), 525–531. doi: 10.1037/0012-1649.25.4.525
- Chu, J. A., Frey, L. M., Ganzel, B. L., & Matthews, J. A. (2000). Memories of childhood abuse: Dissociation, amnesia, and corroboration. *American Journal of Psychiatry*, 156, 749–755.
- Ciechanowski, P. S., Walker, E. A., Katon, W. J., & Russo, J. E. (2002). Attachment theory: a model for health care utilization and somatization. [Research Support, US Gov't, P H S]. *Psychosomatic Medicine*, 64(4), 660–667.
- Cloninger, R. C., Martin, R. L., Guze, S. B., & Clayton, P. L. (1986). A prospective follow-up and family study of somatization in men and women. *American Journal of Psychiatry*, 143, 713–714.
- Cooper, M. S., & Clark, V. P. (2013). Neuroinflammation, neuroautoimmunity, and the co-morbidities of complex regional pain syndrome. *Journal of Neuroimmune Pharmacology*, 8, 452–469.
- Dalenberg, C. J. (2006). Recovered memory and the Daubert criteria: recovered memory as professionally tested, peer reviewed, and accepted in the relevant scientific community. *Trauma, Violence, & Abuse*, 7(4), 274–310. doi: 10.1177/1524838006294572
- Dalenberg, C. J., Brand, B. L., Gleaves, D. H., Dorahy, M. J., Loewenstein, R. J., Cardeña, E., . . . Spiegel, D. (2012). Evaluation of the evidence for the trauma and fantasy models of dissociation. *Psychological Bulletin*, 138(3), 550–588. doi: 10.1037/a0027447
- Dalenberg, C. J., Brand, B. L., Loewenstein, R. J., Gleaves, D. H., Dorahy, M. J., Cardeña, E., . . . Spiegel, D. (2014). Reality versus fantasy: Reply to Lynn et al. (2014). *Psychological Bulletin*, 140(3), 911–920. doi: http://dx.doi.org/10.1037/a0036685
- deCharms, R. C., Maeda, F., Glover, G. H., Ludlow, D., Pauly, J. M., Soneji, D., et al. (2005). Control over brain activation and pain learned by using real-time functional MRI. *Proceedings of the National Academy of Sciences of the United States of America*, 102, 18626–18631.
- Dell, P. F. (2006). A new model of dissociative identity disorder. *Psychiatric Clinics of North America*, 29, 1–26, vii.
- Dell, P. F. (2009). The long struggle to diagnose multiple personality disorder (MPD): Partial MPD. In P. F. Dell & J. A. O'Neil (Eds.), *Dissociation and the dissociative disorders: DSM-VDSM-5 and beyond* (pp. 403–428). Hoboken: Taylor & Francis.
- Demyttenaere, K., Bruffaerts, R., Posada-Villa, J., Gasquet, I., Kovess, V., Lepine, J. P., et al. (2004). Prevalence, severity, and unmet need for treatment of mental disorders in the World Health Organization World Mental Health Surveys. *Journal of the American Medical Association*, 291, 2581–2590.
- Dorahy, M. J., Brand, B. L., Sar, V., Krüger, C., Stavropoulos, P., Martínez-Taboas, A., Middleton, W. (2014). Dissociative identity disorder: An empirical overview. *The Australian and New Zealand journal of psychiatry*, 48(5), 402–417. doi: 10.1177/0004867414527523
- Drob, S., & Meehan, K. (2000). The diagnosis of Ganser Syndrome in the practice of forensic psychology. *American Journal of Forensic Psychology*, 18(3), 37–62.
- Duggal, S., & Sroufe, L. A. (1998). Recovered memory of childhood sexual trauma: a documented case from a longitudinal study. *Journal of Traumatic Stress*, 11(2), 301–321. doi: 10.1023/a:1024403220769
- Edwards, V. J., Fivush, R., Anda, R. F., Felitti, V. J., & Nordenberg, D. F. (2001). Autobiographical memory disturbances in childhood abuse survivors. *Journal of Aggression, Maltreatment & Trauma*, 4(2), 247–263. doi: 10.1300/J146v04n02\_11
- Ehde, D. M., Dillworth, T. M., & Turner, J. A. (2014). Cognitive-behavioral therapy for individuals with chronic pain: efficacy, innovations, and directions for research. *American Psychologist*, 69, 153–166.
- Elliott, D. M. (1997). Traumatic events: prevalence and delayed recall in the general population. *Journal of consulting and clinical psychology*, 65(5), 811. doi: 10.1037/0022-006x.65.5.811
- Elliott, D. M., & Briere, J. (1995). Posttraumatic stress associated with delayed recall of sexual abuse: A general population study. *Journal of Traumatic Stress*, 8(4), 629–647. doi: 10.1002/jts.2490080407
- Faravelli, C., Salvatori, S., Galassi, F., & Aiazzi, L. (1997). Epidemiology of somatoform disorders: A community survey in Florence. *Social Psychiatry and Psychiatric Epidemiology*, 32, 24–29.
- Fishbain, D. A., Cutler, R., Rosomoff, H. L., & Rosomoff, R. S. (2000). Evidence-based data from animal and human experimental studies on pain relief with antidepressants: A structured review. *Pain Medicine*, 1, 310–316.
- Flor, H. (2014). Psychological pain interventions and neurophysiology: implications for a mechanism-based approach. *American Psychologist*, 69, 188–196.
- Freyd, J. J. (1996). *Betrayal trauma: the logic of forgetting childhood abuse*. Cambridge, Mass: Harvard University Press.
- Freyd, J. J., DePrince, A. P., & Zurborgen, E. L. (2001). Self-reported memory for abuse depends upon victim-perpetrator relationship. *Journal of Trauma & Dissociation*, 2(3), 5–15. doi: 10.1300/J229v02n03\_02
- Gatchel, R. J., Peng, Y. B., Peters, M. L., Fuchs, P. N., & Turk, D. C. (2007). The biopsychosocial approach to chronic pain: Scientific advances and future directions. *Psychological Bulletin*, 133, 581–624.
- Giesbrecht, T., Lynn, S. J., Lilienfeld, S. O., & Merckelbach, H. (2008). Cognitive processes in dissociation: an analysis of core theoretical assumptions. *Psychological Bulletin*, 134(5), 617–647. doi: 10.1037/0033-2909.134.5.617
- Gleaves, D. H. (1996). The sociocognitive model of dissociative identity disorder: A reexamination of the evidence. *Psychological Bulletin*, 120, 42–59.
- Grady, D. (1999). A great pretender now faces the truth of illness, *The New York Times*.
- Greeven, A., van Balkom, A. J. L. M., Visser, S., Merckelbach, J. W., van Rood, Y. R., van Dyck, R., et al. (2007). Cognitive behavior therapy and paroxetine in the treatment of hypochondriasis: A randomized controlled trial. *American Journal of Psychiatry*, 164, 91–99.
- Hacking, I. (1998). *Mad travelers: Reflections on the reality of transient mental illness*. Charlottesville, VA: University Press of Virginia.

- Harden, C. L. (1997). Pseudoseizures and dissociative disorders: a common mechanism involving traumatic experiences. [Case Reports]. *Seizure*, 6(2), 151–155.
- Harding, K. J., Skritskaya, N., Doherty, E., & Fallon, B. A. (2008). Advances in understanding illness anxiety. [Review]. *Current Psychiatry Reports*, 10(4), 311–317.
- Heim, C., Wagner, D., Maloney, E., Papanicolaou, D. A., Solomon, L., Jones, J. F., . . . Reeves, W. C. (2006). Early adverse experience and risk for chronic fatigue syndrome: results from a population-based study. [Comparative Study]. *Archives of General Psychiatry*, 63(11), 1258–1266.
- Heller, A. S., van Reekum, C. M., Schaefer, S. M., Lapate, R. C., Radler, B. T., Ryff, C. D., et al. (2013). Sustained striatal activity predicts eudaimonic well-being and cortisol output. *Psychological Science*, 24, 2191–2200.
- Heller, A. S., van Reekum, C. M., Schaefer, S. M., Lapate, R. C., Radler, B. T., Ryff, C. D., et al. (2013). Sustained striatal activity predicts eudaimonic well-being and cortisol output. *Psychological Science*, 24, 2191–2200.
- Herman, J. L., & Schatzow, E. (1987). Recovery and verification of memories of childhood sexual trauma. *Psychoanalytic Psychology*, 4(1), 1–14. doi: 10.1037/0736-9735.4.1.1
- Holmes, E. A., Brown, R. J., Mansell, W., Fearon, R. P., Hunter, E. C., Frasquilho, F., et al. (2005). Are there two qualitatively distinct forms of dissociation? A review and some clinical implications. *Clinical Psychology Review*, 25, 1–23.
- Hubschmid, M., Aybek, S., Maccaferri, G. E., Chocron, O., Gholamrezaee, M. M., Rossetti, A. O., . . . Berney, A. (2015). Efficacy of brief interdisciplinary psychotherapeutic intervention for motor conversion disorder and nonepileptic attacks. [Comparative Study, Randomized Controlled Trial, Research Support, Non-US Gov't]. *General Hospital Psychiatry*, 37(5), 448–455.
- Hunter, E. C., Sierra, M., & David, A. S. (2004). The epidemiology of depersonalisation and derealisation. A systematic review. *Social Psychiatry and Psychiatric Epidemiology*, 39, 9–18.
- Imbierowicz, K., & Egle, U. T. (2003). Childhood adversities in patients with fibromyalgia and somatoform pain disorder. *European Journal of Pain*, 7(2), 113–119. doi: 10.1016/s1090-3801(02)00072-1
- International Society for the Study of Trauma and Dissociation (ISSTD). (2011). Guidelines for treating dissociative identity disorder in adults, third revision: Summary version. *Journal of Trauma and Dissociation* 12, 188–212.
- Irwin, H. J. (1996). Traumatic childhood events, perceived availability of emotional support, and the development of dissociative tendencies. *Child Abuse & Neglect*, 20(8), 701–707. doi: [http://dx.doi.org/10.1016/0145-2134\(96\)00058-0](http://dx.doi.org/10.1016/0145-2134(96)00058-0)
- Jensen, M. P., & Patterson, D. R. (2014). Hypnotic approaches for chronic pain management: clinical implications of recent research findings. *American Psychologist*, 69, 167–177.
- Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2006). Dissociative disorders among adults in the community, impaired functioning, and axis I and II comorbidity. *Journal of Psychiatric Research*, 40(2), 131–140.
- Jovasevic, V., Corcoran, K. A., Leaderbrand, K., Yamawaki, N., Guedea, A. L., Chen, H. J., . . . Radulovic, J. (2015). GABAergic mechanisms regulated by miR-33 encode state-dependent fear. *Nature Neuroscience*.
- Kaplan, S. (2015, November 24). The blind woman who switched personalities and could suddenly see. *The Washington Post*. Retrieved from <https://www.washingtonpost.com/>
- Kaptschuk, T. J., Kelley, J. M., Conboy, L. A., Davis, R. B., Kerr, C. E., Jacobson, E. E., et al. (2008). Components of placebo effect: Randomised controlled trial in patients with irritable bowel syndrome. *British Medical Journal*, 336, 999–1003.
- Kate, M.-A. (2016a). *Can dissociation explain memory impairment for childhood sexual abuse in a non-clinical sample?* Paper presented at the International Society for the Study of Trauma and Dissociation (ISSTD) 33rd Annual Conference, Park 55 Hotel, San Francisco, California.
- Kate, M.-A. (2016b). *Examining the link between parent–child dynamics and experiences of maltreatment during childhood and dissociative experiences and attachment style in adulthood in a nonclinical sample.* Paper presented at the International Society for the Study of Trauma and Dissociation (ISSTD) 33rd Annual Conference, Park 55 Hotel, San Francisco, California.
- Kate, M.-A. (2017). *A review of 80 studies on dissociative experiences and Dissociative Disorders in college populations.* Paper presented at the International Society for the Study of Trauma and Dissociation (ISSTD) 33rd Annual Conference, Crystal Gateway Hotel, Washington DC.
- Katz, J., Rosenbloom, B. N., & Fashler, S. (2015). Chronic pain, psychopathology, and DSM-5 somatic symptom disorder. *Canadian Journal of Psychiatry*, 60(4), 160–167.
- Kihlstrom, J. F., Tatarzyn, D. J., & Holt, I. P. (1993). Dissociative disorders. In P. B. Sutker & H. E. Adams (Eds.), *Comprehensive handbook of psychopathology* (pp. 203–234). New York: Plenum.
- Kirmayer, L. J., Robbins, J. M., & Paris, J. (1994). Somatoform disorders: Personality and social matrix of somatic distress. *Journal of Abnormal Psychology*, 103, 125–136.
- Kisiel, C. L., & Lyons, J. S. (2001). Dissociation as a mediator of psychopathology among sexually abused children and adolescents. *American Journal of Psychiatry*, 158, 1034–1039.
- Koffel, E., & Watson, D. (2009). Unusual sleep experiences, dissociation, and and schizotypy: Evidence for a common domain. *Clinical Psychology Review*, 29(6), 548–559. doi: <http://dx.doi.org/10.1016/j.cpr.2009.06.004>
- Konnopka, A., Schaefer, R., Heinrich, S., Kaufmann, C., Lupp, M., Herzog, W., et al. (2012). Economics of medically unexplained symptoms: A systematic review of the literature. *Psychotherapy and Psychosomatics*, 81, 265–275.
- Kroenke, K., & Swindle, R. (2000). Cognitive-behavioral therapy for somatization and symptom syndromes: a critical review of controlled clinical trials. *Psychotherapy and Psychosomatics*, 69(4), 205–215.

- Lackner, J. M., Gudleski, G. D., & Blanchard, E. B. (2004). Beyond abuse: the association among parenting style, abdominal pain, and somatization in IBS patients. *Behaviour Research and Therapy*, 42(1), 41–56. doi: [http://dx.doi.org/10.1016/S0005-7967\(03\)00069-X](http://dx.doi.org/10.1016/S0005-7967(03)00069-X)
- Larkin, H., Shields, J. J., & Anda, R. F. (2012). The health and social consequences of adverse childhood experiences (ACE) across the lifespan: an introduction to prevention and intervention in the community. [Introductory, Research Support, US Gov't, P H S]. *Journal of Prevention and Intervention in the Community*, 40(4), 263–270.
- Lee, K., Johnson, M. H., Harris, J., & Sundram, F. (2016). The resource utilisation of medically unexplained physical symptoms. *SAGE Open Medicine*, 4, 2050312116666217. doi: 10.1177/2050312116666217
- Lieb, R., Meinlschmidt, G., & Araya, R. (2007). Epidemiology of the association between somatoform disorders and anxiety and depressive disorders: An update. *Psychosomatic Medicine*, 69, 860–863.
- Lilienfeld, S. O., Lynn, S. J., Kirsch, I., Chaves, J. F., Sarbin, T. R., & Ganaway, G. K. (1999). Dissociative identity disorder and the sociogenic model: Recalling lessons from the past. *Psychological Bulletin*, 125, 507–523.
- Looper, K. J., & Kirmayer, L. J. (2002). Behavioral medicine approaches to somatoform disorders. *Journal of Consulting and Clinical Psychology*, 70, 810–827.
- Lynn, S. J., & Rhue, J. W. (1988). Fantasy proneness: hypnosis, developmental antecedents, and psychopathology. *American Psychologist*, 43(1), 35–44. doi: 10.1037/0003-066x.43.1.35
- Lynn, S. J., Lilienfeld, S. O., Merckelbach, H., Giesbrecht, T., McNally, R. J., Loftus, E. F., ... Malaktaris, A. (2014). The trauma model of dissociation: Inconvenient truths and stubborn fictions. Comment on Dalenberg et al. (2012). *Psychological Bulletin*, 140(3), 896–910. doi: <http://dx.doi.org/10.1037/a0035570>
- Lynn, S. J., Lilienfeld, S. O., Merckelbach, H., Giesbrecht, T., & van der Kloet, D. (2012). Dissociation and dissociative disorders: challenging conventional wisdom. *Current Directions in Psychological Science*, 21(1), 48–53. doi: 10.1177/0963721411429457
- Lyons-Ruth, K. (2003). Dissociation and the parent-infant dialogue: a longitudinal perspective from attachment research. *Journal of the American Psychoanalytic Association*, 51(3), 883–911. doi: 10.1177/00030651030510031501
- Maaranen, P., Tanskanen, A., Haatainen, K., Koivumaa-Honkanen, H., Hintikka, J., & Viinamäki, H. (2004). Somatoform dissociation and adverse childhood experiences in the general population. [Comparative Study]. *Journal of Nervous and Mental Disease*, 192(5), 337–342.
- Main, M., & Hesse, E. (1990). Parents' unresolved traumatic experiences are related to infant disorganized attachment status: is frightened and/or frightening parental behavior the linking mechanism? . In M. T. Greenberg, D. Cicchetti & E. M. Cummings (Eds.), *Attachment in the preschool years: Theory, research, and intervention* (pp. 161–182). Chicago: University of Chicago Press.
- Main, M., & Morgan, H. J. (1996). Disorganization and disorientation in infant strange situation behavior: Phenotypic resemblance to dissociative states. In L. Michelson & W. J. Ray (Eds.), *Handbook of dissociation: theoretical, empirical, and clinical perspectives* (pp. 107–138). New York: Plenum Press.
- Maldonado, J. R., Butler, L. D., & Spiegel, D. (1998). Treatments for dissociative disorders. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 423–446). New York: Oxford University Press.
- Marcus, D. K., Gurley, J. R., Marchi, M. M., & Bauer, C. (2007). Cognitive and perceptual variables in hypochondriasis and health anxiety: A systematic review. *Clinical Psychology Review*, 27, 127–139.
- Marquez, M., Segui, J., Garcia, L., Canet, J., & Ortiz, M. (2001). Is panic disorder with psychosensorial symptoms (depersonalization-derealization) a more severe clinical subtype? *Journal of Nervous and Mental Disease*, 189, 332–335.
- Martin, A., & Jacobi, F. (2006). Features of hypochondriasis and illness worry in the general population in Germany. *Psychosomatic Medicine*, 68, 770–777.
- Mayer, E. A., Berman, S., Suyenobu, B., Labus, J., Mandelkern, M. A., Naliboff, B. D., et al. (2005). Differences in brain responses to visceral pain between patients with irritable bowel syndrome and ulcerative colitis. *Pain*, 115, 398–409.
- Mayo, J. P., Jr., & Haggerty, J. J., Jr. (1984). Long-term psychotherapy of Munchausen syndrome. [Case Reports]. *American Journal of Psychotherapy*, 38(4), 571–578.
- McCracken, L. M., & Vowles, K. E. (2014). Acceptance and commitment therapy and mindfulness for chronic pain: model, process, and progress. *American Psychologist*, 69, 178–187.
- McManus, F., Surawy, C., Muse, K., Vazquez-Montes, M., & Williams, J. M. (2012). A randomized clinical trial of mindfulness-based cognitive therapy versus unrestricted services for health anxiety (hypochondriasis). *Journal of Consulting and Clinical Psychology*, 80, 817–828.
- Mendis, S., & Hodgson, R. E. (2012). Ganser syndrome: examining the aetiological debate through a systematic case report review. *The European Journal of Psychiatry*, 26. <http://dx.doi.org/10.4321/S0213-61632012000200003>
- Mezzich, J. E., Fabrega, H., Jr., Coffman, G. A., & Haley, R. (1989). DSM-III disorders in a large sample of psychiatric patients: Frequency and specificity of diagnoses. *American Journal of Psychiatry*, 146, 212–219.
- Middleton, W. (2004a). Anna O and Hanna Q. [Case Reports, Comment]. *Australasian Psychiatry*, 12(3), 239–244.
- Middleton, W. (2004b). Dissociative disorders: a personal 'work in progress'. *Australasian Psychiatry*, 12(3), 245–252.
- Middleton, W. (2013). Ongoing incestuous abuse during adulthood. *Journal of Trauma and Dissociation*, 14(3), 251–272.
- Middleton, W. (2014). Parent-child incest that extends into adulthood: a survey of international press reports, 2007–2012. In V. Sar, W. Middleton & M. Dorahy (Eds.), *Global Perspectives on Dissociative Disorders: Individual and Societal Oppression* (pp. 45–64). London: Routledge (Taylor and Francis).
- Middleton, W., & Butler, J. (1998). Dissociative identity disorder: an Australian series. *Australian and New Zealand Journal of Psychiatry*, 32(6), 794–804.

- Middleton, W., De Marni Cromer, L., & Freyd, J. J. (2005). Remembering the past, anticipating a future. [Review]. *Australasian Psychiatry*, 13(3), 223–233.
- Moormann, P. M., Albach, F., & Bermond, B. (2012). Do alexithymia, dissociation, and CSA explain the controversial topic of memory recovery? In E. A. Kalfoglou (Ed.), *Sexual abuse — breaking the silence*. InTech.
- Morley, S. (1997). Pain management. In A. Baum, S. Newman, J. Weinman, R. West & C. McManus (Eds.), *Cambridge handbook of psychology, health and medicine* (pp. 234–237). Cambridge, UK: Cambridge University Press.
- Mueller-Pfeiffer, C., Rufibach, K., Perron, N., Wyss, D., Kuenzler, C., Prezewowsky, C., et al. (2012). Global functioning and disability in dissociative disorders. *Psychiatry Research*, 200, 475–481.
- Muse, K., McManus, F., Hackmann, A., & Williams, M. (2010). Intrusive imagery in severe health anxiety: Prevalence, nature and links with memories and maintenance cycles. *Behaviour Research and Therapy*, 48, 792–798.
- Nathan, D. (2011). *Sybil exposed: The extraordinary story behind the famous multiple personality case*. New York: Free Press.
- Nilsen, W. J. (2000). *The relationship between trauma and dissociation: Is the development of dissociative symptoms mediated by family functioning?* (PhD), Purdue University.
- Noyes, R. (1999). The relationship of hypochondriasis to anxiety disorders. *General Hospital Psychiatry*, 21, 8–17.
- Ogawa, J. R., Sroufe, L. A., Weinfield, N. S., Carlson, E. A., & Egeland, B. (1997). Development and the fragmented self: Longitudinal study of dissociative symptomatology in a nonclinical sample. *Development and Psychopathology*, 9(4), 855–879. doi: 10.1017/s0954579497001478
- Park, S. H., Videlock, E. J., Shih, W., Presson, A. P., Mayer, E. A., & Chang, L. (2016). Adverse childhood experiences are associated with irritable bowel syndrome and gastrointestinal symptom severity. *Journal of Neurogastroenterology and Motility*, 28(8), 1252–1260.
- Payne, A., & Blanchard, E. B. (1995). A controlled comparison of cognitive therapy and self-help support groups in the treatment of irritable bowel syndrome. *Journal of Consulting and Clinical Psychology*, 63, 779–786.
- Perry, B. D. (2003). *Effects of traumatic events on children: an introduction*. Houston, Texas: The Child Trauma Academy.
- Persing, J. S., Stuart, S. P., Noyes, R., & Happel, R. L. (2000). Hypochondriasis: The patient's perspective. *International Journal of Psychiatry in Medicine*, 30, 329–342.
- Phillips, M. R., Li, X., & Zhang, Y. (2002). Suicide rates in China, 1995–99. *The Lancet*, 359, 835–840.
- Pope, H. G. J., Poliakoff, M. B., Parker, M. P., Boynes, M., & Hudson, J. J. (2006). Is dissociative amnesia a culture-bound syndrome? Findings from a survey of historical literature. *Psychological Medicine*, 37, 1067–1068.
- Porges, S. W. (2011). *The polyvagal theory: Neurophysiological foundations of emotions, attachment, communication and self-regulation*. New York: Norton.
- Porges, S. W. (2013). *The Polyvagal Theory; Implications for Understanding and Treating Trauma and Dissociation* Paper presented at the International Society for the Study of Trauma and Dissociation (ISSTD) 30th Annual Conference, Hilton Hotel, Baltimore.
- Porter, S., Yuille, J. C., & Lehman, D. R. (1999). The nature of real, implanted, and fabricated memories for emotional childhood events: implications for the recovered memory debate. [Research Support, Non-US Gov't]. *Law Hum Behav*, 23(5), 517–537.
- Preston, N. (2013). *After Sybil: from the letters of Shirley Mason*. Conshohocken, Pennsylvania: Infinity Publishing.
- Price, D. D., Craggs, J. G., Zhou, Q., Verne, G. N., Perlstein, W. M., & Robinson, M. E. (2009). Widespread hyperalgesia in irritable bowel syndrome is dynamically maintained by tonic visceral impulse input and placebo/nocebo factors: Evidence from human psychophysics, animal models, and neuroimaging. *NeuroImage*, 47, 995–1001.
- Putnam, F. W. (1996). A brief history of multiple personality disorder. *Child and Adolescent Psychiatric Clinics of North America*, 5, 263–271.
- Pymont, C., & Butterworth, P. (2015). Longitudinal cohort study describing persistent frequent attenders in Australian primary healthcare. *BMJ Open*, 5(10). doi: 10.1136/bmjopen-2015-008975
- Rachman, S. (2012). Health anxiety disorders: a cognitive construal. *Behaviour Research and Therapy*, 50, 502–512.
- Reinders, A. A. T. S., Willemsen, A. T. M., Vos, H. P. J., den Boer, J. A., & Nijenhuis, E. R. S. (2012). Fact or factitious? A psychobiological study of authentic and simulated dissociative identity states. *PLoS ONE*, 7(6), e39279. doi: 10.1371/journal.pone.0039279
- Reinhold, N., & Markowitsch, H. J. (2009). Retrograde episodic memory and emotion: a perspective from patients with dissociative amnesia. *Neuropsychologia*, 47, 2197–2206.
- Rief, W., & Auer, C. (2001). Is somatization a habituation disorder? Physiological reactivity in somatization syndrome. *Psychiatry Research*, 101, 63–74.
- Rief, W., & Broadbent, E. (2007). Explaining medically unexplained symptoms-models and mechanisms. *Clinical Psychology Review*, 27, 821–841.
- Rief, W., Hessel, A., & Braehler, E. (2001). Somatization symptoms and hypochondriacal features in the general population. *Psychosomatic Medicine*, 63(4), 595–602.
- Rief, W., Buhlmann, U., Wilhelm, S., Borkenhagen, A., & Brähler, E. (2006). The prevalence of body dysmorphic disorder: A population-based survey. *Psychological Medicine*, 36, 877–885.
- Rodewald, F., Wilhelm-Goling, C., Emrich, H. M., Reddemann, L., & Gast, U. (2011). Axis-I comorbidity in female patients with dissociative identity disorder and dissociative identity disorder not otherwise specified. *Journal of Nervous and Mental Disease*, 199, 122–131.
- Ross, C. A. (1991). Epidemiology of multiple personality disorder and dissociation. *Psychiatric Clinics of North America*, 14, 503–517.

- Ross, C. A. (1997). *Dissociative identity disorder: diagnosis, clinical features, and treatment of multiple personality* (2nd ed.). New York: Wiley.
- Ross, C. A., Duffy, C. M. M., & Ellason, J. W. (2002). Prevalence, reliability and validity of dissociative disorders in an inpatient setting. *Journal of Trauma and Dissociation*, 3, 7–17.
- Ross, C. A., Heber, S., Norton, G. R., Anderson, D., Anderson, G., & Barchet, P. (1989). The Dissociative Disorders Interview Schedule: A structured interview. *Dissociation*, 2(3), 169–189.
- Ross, C. A., & Ness, L. (2010). Symptom patterns in dissociative identity disorder patients and the general population. *Journal of Trauma and Dissociation*, 11(3), 450–68.
- Royal Commission into Institutional Responses to Child Sexual Abuse. (2015). *Report of Case Study No. 17. The response of the Australian Indigenous Ministries, the Australian and Northern Territory governments and the Northern Territory police force and prosecuting authorities to allegations of child sexual abuse which occurred at the Retta Dixon Home, by Commissioners R. Fitzgerald and H. Milroy*. Canberra: Government of Australia.
- Sar, V. (2011). Epidemiology of dissociative disorders: an overview. *Epidemiology Research International*, 2011. doi: 10.1155/2011/404538
- Sar, V., Akyuz, G., & Dogan, O. (2007). Prevalence of dissociative disorders among women in the general population. *Psychiatry Research*, 149(1–3), 169–176. doi: 10.1016/j.psychres.2006.01.005
- Sar, V., Akyuz, G., Kundakci, T., Kiziltan, E., & Dogan, O. (2004). Childhood trauma, dissociation, and psychiatric comorbidity in patients with conversion disorder. *American Journal of Psychiatry*, 161, 2271–2276.
- Schefflin, A. W., & Brown, D. (1996). Repressed memory or dissociative amnesia: what the science says. *Journal of Psychiatry & Law*, 24(2), 143.
- Schlumpf, Y. R., Reinders, A. A. T. S., Nijenhuis, E. R. S., Luechinger, R., van Osch, M. J. P., & Jäncke, L. (2014). Dissociative part-dependent resting-state activity in dissociative identity disorder: a controlled fMRI perfusion study. *PLoS ONE*, 9(6), e98795. doi: 10.1371/journal.pone.0098795
- Schreiber, F. L. (1973). *Sybil*. Chicago: Regnery.
- Scroppo, J. C., Drob, S. L., Weinberger, J. L., & Eagle, P. (1998). Identifying dissociative identity disorder: A selfreport and projective study. *Journal of Abnormal Psychology*, 107, 272–284.
- Siegel, D. (2014). *The developing mind: how relationships and the brain interact to shape who we are* (2nd ed.). New York: The Guilford Press.
- Sierra, M., & Berrios, G. E. (2000). The Cambridge depersonalization scale: a new instrument for the measurement of depersonalization. *Psychiatry Research*, 93. doi: 10.1016/s0165-1781(00)00100-1
- Silberg, J. L. (2004). Guidelines for the evaluation and treatment of dissociative symptoms in children and adolescents. *Journal of Trauma & Dissociation*, 5(3), 119–150.
- Simeon, D. (2009). Depersonalization disorder. In P. F. Dell & J. A. O'Neil (Eds.), *Dissociation and the dissociative disorders: DSM-VDSM-5 and beyond* (pp. 435–444). Hoboken: Taylor & Francis.
- Simeon, D., Gross, S., Guralnik, O., Stein, D. J., Schmeidler, J., & Hollander, E. (1997). Feeling unreal: 30 cases of DSM-III-R depersonalization disorder. *American Journal of Psychiatry*, 154, 1107–1112.
- Simeon, D., Guralnik, O., Schmeidler, J., Sirof, B., & Knutelska, M. (2001). The role of childhood interpersonal trauma in depersonalization disorder. *The American Journal of Psychiatry*, 158(7), 1027–1033. doi: 10.1176/appi.ajp.158.7.1027
- Simeon, D., Knutelska, M., Nelson, D., & Guralnik, O. (2003). Feeling unreal: a depersonalization disorder update of 117 cases. [Research Support, Non-US Gov't, Research Support, US Gov't, P H S]. *J Clin Psychiatry*, 64(9), 990–997.
- Simon, G. E. (1998). Management of somatoform and factitious disorders. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 408–422). New York: Oxford University Press.
- Simon, G. E., Gureje, O., & Fullerton, C. (2001). Course of hypochondriasis in an international primary care study. *General Hospital Psychiatry*, 23, 51–55.
- Simon, G. E., Von Korff, M., Piccinelli, M., Fullerton, C., & Ormel, J. (1999). An international study of the relation between somatic symptoms and depression. *New England Journal of Medicine*, 341, 1329–1335.
- Smith, G. (1992). The epidemiology and treatment of depression when it coincides with somatoform disorders, somatization, or panic. *General Hospital Psychiatry*, 14, 265–272.
- Somer, E., Amos-Williams, T., & Stein, D. J. (2013). Evidence-based treatment for Depersonalisation-derealisation Disorder (DPRD). *BMC Psychology*, 1(1), 20. doi: 10.1186/2050-7283-1-20
- Spanos, N. P. (1994). Multiple identity enactments and multiple personality disorder: A sociocognitive perspective. *Psychological Bulletin*, 116, 143–165.
- Staniloiu, A., & Markowitsch, H. J. (2014). Dissociative amnesia. *The Lancet Psychiatry*, 1(3), 226–241.
- Steinberg, M. (1994). *Interviewer's guide to the Structured Clinical Interview for DSM-IV® Dissociative Disorders (SCID-D)*. Arlington, VA: American Psychiatric Association.
- Steinberg, M., & Schnall, M. (2001). *The stranger in the mirror*. New York: Quill.
- Stone, J., Smyth, R., Carson, A., Lewis, S., Prescott, R., Warlow, C., et al. (2005). Systematic review of misdiagnosis of conversion symptoms and "hysteria". *British Medical Journal*, 331, 989.
- Stone, J., LaFrance, W. C., Jr., Levenson, J. L., & Sharpe, M. (2010). Issues for DSM-5: Conversion disorder. *American Journal of Psychiatry*, 167, 626–627.

- Streltzer, J., & Johansen, L. G. (2006). Prescription drug dependence and evolving beliefs about chronic pain management. *American Journal of Psychiatry*, 163, 594–598.
- Sutcliffe, J. P., & Jones, J. (1962). Personal identity, multiple personality, and hypnosis. *International Journal of Clinical and Experimental Hypnosis*, 10, 231–269.
- Thomson, A. B., & Page, L. A. (2007). Psychotherapies for hypochondriasis. *Cochrane Database of Systematic Reviews*, Issue 4, CD006520.
- Trask, P. C., & Sigmon, S. T. (1997). Munchausen syndrome: a review and new conceptualization. *Clinical Psychology: Science and Practice*, 4(4), 346–358. doi: 10.1111/j.1468-2850.1997.tb00126.x
- Uchinuma, Y., & Sekine, Y. (2000). Dissociative identity disorder (DID) in Japan: A forensic case report and the recent increase in reports of DID. *International Journal of Psychiatry in Clinical Practice*, 4, 155–160.
- Vaillant, G. E. (2011). Involuntary coping mechanisms: a psychodynamic perspective. *Dialogues in Clinical Neuroscience*, 13(3), 366–370.
- van der Hart, O., Lierens, R., & Goodwin, J. (1996). Jeanne Fery: a sixteenth-century case of dissociative identity disorder. *Journal of Psychohistory*, 24(1), 18–35.
- van der Kolk, B. (2014). *The body keeps the score: Brain, mind, and body in the healing of trauma*. New York: Viking.
- Veehof, M. M., Oskam, M. J., Schreurs, K. M., & Bohlmeijer, E. T. (2011). Acceptance-based interventions for the treatment of chronic pain: a systematic review and meta-analysis. *Pain*, 152, 533–542.
- Villemure, C., & Bushnell, M. C. (2009). Mood influences supraspinal pain processing separately from attention. *Journal of Neuroscience* 29, 705–715.
- Waller, N. G., Putnam, F. W., & Carlson, E. B. (1996). Types of dissociation and dissociative types: a taxometric analysis of dissociative experiences. *Psychological Methods*, 1(3), 300–321. doi: 10.1037/1082-989x.1.3.300
- Warwick, H. M. C., & Salkovskis, P. M. (2001). Cognitive-behavioral treatment of hypochondriasis. In D. R. Lipsitt & V. Starcevic (Eds.), *Hypochondriasis: Modern perspectives on an ancient malady* (pp. 314–328). London: Oxford University Press.
- Welburn, K. R., Fraser, G. A., Jordan, S. A., Cameron, C., Webb, L. M., & Raine, D. (2003). Discriminating dissociative identity disorder from schizophrenia and feigned dissociation on psychological tests and structured interview. *Journal of Trauma & Dissociation*, 4(2), 109–130. doi: 10.1300/J229v04n02\_07
- Wiech, K., & Tracey, I. (2009). The influence of negative emotions on pain: behavioral effects and neural mechanisms. *NeuroImage*, 47, 987–994.
- Williams, L. M. (1994). Recall of childhood trauma: a prospective study of women's memories of child sexual abuse. *Journal of consulting and clinical psychology*, 62(6), 1167–1176. doi: 10.1037/0022-006x.62.6.1167
- Williams, L. M. (1995). Recovered memories of abuse in women with documented child sexual victimization histories. *Journal of traumatic stress*, 8(4), 649–673. doi: 10.1002/jts.2490080408
- Wilsnack, S. C., Wilsnack, R. W., Wonderlich, S. A., Kristjanson, A. F., & Vogeltanz-Holm, N. D. (2002). Self-reports of forgetting and remembering childhood sexual abuse in a nationally representative sample of US women. *Child Abuse & Neglect*, 26(2), 139–147. doi: 10.1016/s0145-2134(01)00313-1
- Winnicott, D. W. (1971). *Playing and reality*. London: Tavistock.

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## CHAPTER 6

# Schizophrenia

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 6.1** describe the clinical symptoms of schizophrenia, including positive, negative and disorganised symptoms
  - 6.2** differentiate the genetic factors, both behavioural and molecular, in the aetiology of schizophrenia
  - 6.3** describe how the brain has been implicated in schizophrenia
  - 6.4** describe the role of environmental factors in the aetiology and relapse of schizophrenia
  - 6.5** discuss the appropriate use of medication and psychological treatments for schizophrenia.
-

## OPENING SCENARIO

I have just passed my 10-year anniversary since being diagnosed with schizophrenia. Where has the time gone? Some battles are over after one skirmish, others last a lifetime.

When my diagnosis came, my life was full of paranoia, delusions, and I was out of control. I reacted to every overpowering influence with bizarre behaviour. The voices kept me isolated, on guard even when I was trying to sleep. I was in control of a world that no one else could see or hear. In truth, I was on a one-way street that would only lead to my demise.

The last 10 years has not been easy. There have been hospital stays, numerous medications changes, and major lifestyle changes. All of that has brought me to today where I am in a state of constant recovery. I am fully aware that recovery never ends. As simple as it may sound: I must follow the rules of recovery. Not for the next month or six months, but for the rest of my life. About those rules of recovery . . .

Rule 1 — Stay on your medication. You may start to feel better after a month or two, but that does not mean you can go off your medication. You are not 'healed'. The medication is making a difference. NEVER make a medication change without talking to your doctor.

Rule 2 — Find your support system. I have been fortunate to have great family support, but I understand this is not always the case. There are mental health professionals at McGuire who can guide you to finding your support system. Church groups, neighbours, and your fellow veterans are all places where you will find people are on your side and will give you the support you need to continue your recovery. You cannot do this alone; you must ask for help.

Rule 3 — Find something you enjoy doing. This activity will shield you from the troubling thoughts that get into your brain. Some examples are: music, brain game, writing in a journal, exercise, following a sport or a team. Use a computer to virtually visit the places you served while you were on active duty. Stay current by watching the news and other informative shows such as the History or National Geographic Channel.

Rule 4 — Stay away from habits that might be harmful to your medication or your health. When I first began taking my medication, I thought alcohol would keep the voices away. I was wrong! I had to totally quit. With the help of God, I was also able to quit my three-pack-a-day cigarette habit.

Illness, mediation, and recovery is a battle. The four weapons that I have mentioned above will give you a better chance of winning the recovery battle. As a veteran you know what fighting a battle means. It is not easy, but ongoing victory is possible. In the 10 years since my first diagnosis, I have fought homelessness, alcoholism, isolation, and at times hopelessness, but after 10 years, I know I am here to defeat my illness. I am not defined by my illness. I am Jason Jepson. I have schizophrenia, and I am recovering.

*Source:* Jepson (2016).

## QUESTIONS

1. What is the difference between cure and recovery?
2. Jepson describes the importance of developing a support network. What are the advantages of a support network over and above contact with mental health professionals? Are there any disadvantages?

## Introduction

The young man described in the opening scenario was diagnosed with schizophrenia. **Schizophrenia** is a disorder characterised by faulty perception and beliefs; a change in levels of motivation and emotional expressiveness; disordered thinking and behaviour; and changes in cognition. There are a wide range of clinical manifestations, however, it is common for people with the disorder to withdraw from other people and from everyday reality, often into a life of odd beliefs (delusions) and hallucinations. Despite the effort that has been placed in describing and investigating schizophrenia it has been difficult to uncover the causes of the disorder and develop effective methods to treat it. We still have a long way to go before we fully understand the multiple factors that trigger schizophrenia and develop treatments that are both effective and free of unpleasant adverse effects.

The symptoms of schizophrenia invade every aspect of a person's life: the way someone thinks, feels and behaves. Not surprisingly then, these symptoms can interfere with maintaining stable employment, living independently and having close relationships with other people. This leads to significant levels of

disability, with just 21.5 percent being employed over a 12-month period and 85 percent being dependent upon government welfare payments for their livelihood (Morgan et al., 2011); only 17.1 percent of people with a psychotic illness, the most severe of which is schizophrenia, were living with a partner (only 12 percent for males) as against 61 percent of the general population; and nearly 38 percent had experienced stigma or persecution from other people in the past year (Morgan et al., 2011). Substance use rates are high with 63.2 percent of males and 41.7 percent of females having a lifetime history of abusing or being dependent upon cannabis or other illicit drugs (Morgan et al., 2011). A similar proportion abused alcohol. This level of abuse may reflect an attempt to achieve some relief from the symptoms (Blanchard et al., 1999), however, it does also contribute to the risk of relapse as well as the level of disability. About 5 percent of people with schizophrenia will end their own life by suicide, usually in the first few years of having the illness (Palmer, Pankratz, & Bostwick, 2005). People with schizophrenia are also more likely to die from any cause than people in the general population (Saha, Chant, & McGrath, 2007) and their mortality rates are as high or higher than the rates for people who smoke (Chesney, Goodwin, & Fazel, 2014). In Australia, people with a psychotic illness have very high rates of diabetes mellitus type 2, hypertension and metabolic syndrome, which, when combined with their high rates of smoking and unhealthy lifestyle, greatly increase the risk of premature death (Galletly et al., 2012).

The lifetime morbid risk of developing schizophrenia is around 7 in 1000, and it is no more common in men than women (Saha, Chant, Welham, & McGrath, 2005). It is more common in urban settings and in people who have migrated (Saha et al., 2005). Schizophrenia sometimes begins in childhood, but it usually appears in late adolescence or early adulthood and usually presents somewhat earlier in men than in women. People with schizophrenia typically have a number of acute episodes of their symptoms early in the course of their illness and less severe but still debilitating symptoms between episodes.

## 6.1 Clinical descriptions of schizophrenia

**LEARNING OUTCOME 6.1** Describe the clinical symptoms of schizophrenia, including positive, negative and disorganised symptoms.

The range of symptoms in the diagnosis of schizophrenia is extensive, although people with the disorder typically have only some of these symptoms at any given time (see the DSM-5 criteria box). It is possible to diagnose two different people with schizophrenia with virtually no symptom overlap. Schizophrenia symptoms can be grouped in a number of ways, but are perhaps best described in five broad domains: positive, negative, disorganised, cognitive and mood/anxiety. Table 6.1 shows the symptoms that comprise these domains. In the following sections, we describe in some detail the individual symptoms that make up these domains.

### DSM-5

#### DSM-5 criteria for schizophrenia

- Two or more of the following symptoms for at least one month; one symptom should be either 1, 2 or 3:
  - (1) delusions
  - (2) hallucinations
  - (3) disorganised speech
  - (4) disorganised (or catatonic) behaviour
  - (5) negative symptoms (diminished motivation or emotional expression).
- Functioning in work, relationships or self-care has declined since onset.
- Signs of disorder for at least six months; or, if during a prodromal or residual phase, negative symptoms or two or more of symptoms 1–4 in less severe form.

### Positive symptoms

**Positive symptoms** comprise excesses and distortions, and include hallucinations and delusions. For the most part, acute episodes of schizophrenia are characterised by positive symptoms.

**TABLE 6.1** Summary of the major symptom domains in schizophrenia

Positive symptoms	Negative symptoms	Disorganised symptoms	Cognitive	Mood/anxiety
Delusions, hallucinations	Avolition, alogia, anhedonia, blunted affect, asociality	Disorganised behaviour, disorganised speech	Attention and concentration, memory, speed of processing, executive functioning Social cognition	Anxiety, obsessions and compulsions, depression

## Delusions

No doubt all of us at one time or another have believed that others thought ill of us. Some of the time this belief may be justified. After all, who is universally loved? Consider, though, the anguish you would feel if you were firmly convinced that many people did not like you — indeed, that they disliked you so much that they were plotting against you. Imagine that your persecutors have sophisticated listening devices that let them tune in on your most private conversations and gather evidence in a plot to discredit you. Those around you, including your loved ones, are unable to reassure you that people are not spying on you. Even your closest friends are gradually joining forces with your tormentor. Anxious and angry, you begin taking counteractions against the persecutors. You carefully check any new room you enter for listening devices. When you meet people for the first time, you question them at great length to determine whether they are part of the plot against you.

Such **delusions**, which are beliefs contrary to reality and firmly held in spite of disconfirming evidence, are common positive symptoms of schizophrenia. Persecutory delusions such as those just described were found in 65 percent of a large, cross-national sample of people diagnosed with schizophrenia (Sartorius, Shapiro, & Jablonksy, 1974). Delusions may take several other forms as well, including the following.

- A person may believe that thoughts that are not his or her own have been placed in his or her mind by an external source (*thought insertion*) or have been taken from their minds (*thought withdrawal*). Together, these phenomena are sometimes called *thought alienation*.
- A person may believe that his or her thoughts are broadcast or transmitted, so that others know what he or she is thinking; this is called *thought broadcasting*. For example, when walking down the street, a man may look suspiciously at passers-by, thinking that they are able to hear what he is thinking even though he is not saying anything out loud. This is sometimes explained as telepathy or ESP.

Believing that others are taking special notice is a common paranoid delusion.



- A person may believe that an external force controls his or her feelings or behaviours. For example, a person may believe that his or her behaviour is being controlled by the signals emitted from mobile telephone towers.
- A person may have **grandiose delusions**, an exaggerated sense of his or her own importance, power, knowledge or identity. For example, a woman may believe that she can cause the wind to change direction just by moving her hands.
- A person may have **ideas of reference**, incorporating unimportant events within a delusional framework and reading personal significance into the trivial activities of others. For instance, people with this symptom might think that overheard segments of conversations are about them, that the frequent appearance of the same person on a street where they customarily walk means that they are being watched, and that what they see on television or read in magazines somehow refers to them.
- A person may believe that the people around them have been replaced by imposters, that their parents are not their parents, and the doctors and nurses that they might be treated by are not real health professionals. A variant of this is that someone, usually persecuting the individual, is changing their appearance. These delusional ideas are known collectively as *misidentification delusions*.

Although delusions are found among more than half of people with schizophrenia, they are also found among people with other diagnoses, including bipolar disorder, major depressive disorder with psychotic features and delusional disorder.

## Hallucinations and other disturbances of perception

People with schizophrenia frequently report that the world seems somehow different or even unreal to them. This can be due to depersonalisation or derealisation. For other people it can form part of a continuum of poorly diagnosed psychotic symptoms that place them at risk of developing a psychotic illness (see focus on discovery 6.3). Others report difficulty making sense of what is happening around them:

I can't concentrate on television because I can't watch the screen and listen to what is being said at the same time. I can't seem to take in two things like this at the same time especially when one of them means watching and the other means listening. On the other hand I seem to be always taking in too much at the one time and then I can't handle it and can't make sense of it. (McGhie & Chapman, 1961, p. 106)

The most dramatic distortions of perception are **hallucinations** — sensory experiences in the absence of any relevant stimulation from the environment. They are more often auditory than visual; 74 percent of one sample of people with schizophrenia reported having auditory hallucinations (Sartorius et al., 1974). However, hallucinations can occur in any of the five sensory modalities.

Some people with schizophrenia report hearing their own thoughts spoken by another voice. Other people may claim that they hear voices arguing, and others hear voices commenting on their behaviour. Many people with schizophrenia experience their hallucinations as frightening or annoying. In one study of nearly 200 people with schizophrenia, those who had hallucinations that were longer, louder, more frequent and experienced in the third person found them unpleasant. Hallucinations that were believed to come from a known person were experienced more positively (Copolov, Mackinnon, & Trauer, 2004).

Some theorists propose that people who have auditory hallucinations misattribute their own voice as being someone else's voice. Behavioural studies have shown that people with hallucinations are more likely to misattribute recordings of their own speech to a different source than are people without hallucinations or healthy controls (Allen et al., 2004). Neuroimaging studies have examined what happens in the brain during auditory hallucinations. These studies have found greater activity in Broca's area, an area of the frontal cortex that supports our ability to produce speech, and in Wernicke's area, an area of the temporal cortex that supports our ability to understand speech, when people with schizophrenia report hearing voices (Curcio-Blake et al., 2013). A meta-analysis of 10 neuroimaging studies found strongest activation in those areas of the brain associated with speech production (e.g., Broca's area), but it also found activation in areas associated with speech processing and understanding in the temporal lobes (Jardri, Pouchet, Pins, & Thomas, 2011). One suggestion that unifies these findings is the possibility that people who experience auditory hallucinations fail to accurately predict what sensations should arise from their own actions in a

failure of the corollary discharge mechanisms in the brain — the mechanism that unconsciously monitors and matches the results of self-generated actions (Whitford, Ford, Mathalon, Kubicki, & Shenton, 2012).

Delusions and hallucinations, though frequent in schizophrenia, are not specific to schizophrenia and can occur in other psychological disorders, including bipolar disorder, major depressive disorder and substance use disorders, to name just a few. Nor are they essential for the diagnosis. Someone presenting with these symptoms may be incorrectly given a diagnosis of schizophrenia. In focus on discovery 6.1, we discuss the case of a young woman who had these symptoms but who most definitely did not have schizophrenia. This case points to the importance of conducting thorough assessments, as we discussed in an earlier chapter.

#### FOCUS ON DISCOVERY 6.1

##### An example of misdiagnosis

A successful young woman who is a reporter for a major city newspaper begins to feel inklings of paranoia. At first, it doesn't seem like paranoia, but more like jealousy. She looks through her boyfriend's drawers for evidence he is cheating on her — something not atypical for new relationships.

But later, she begins to think people are taking special notice of her; that she is receiving special messages from TV; that her family is plotting against her. People are talking about her, whispering her name. People are spying on her and trying to hurt her. When others don't take her concerns seriously, she erupts into rages and becomes very agitated and then without warning, begins sobbing uncontrollably.

She reports that she sees bright colours; then she reports having visions and seeing things that are most definitely not observed by others. In short, she begins to have visual hallucinations.

She begins to feel like she is having an out-of-body experience, like she is looking down on herself from above, watching her every move. She writes down random musings in a journal, but the thoughts are not connected and are very disorganised. Then, she has a seizure.

What can account for these symptoms, including hallucinations, paranoid delusions, disorganised thinking and behaviour; and rapidly changing moods? Is it schizophrenia? Bipolar disorder with psychotic features? A substance use disorder? A seizure disorder? All of these are plausible diagnoses for the symptoms this young woman presented with. Yet none of them was correct.

In her beautifully written book, *Brain on Fire: My Month of Madness*, Susannah Cahalan writes about her experience with these symptoms over the course of a month. She did not remember many of these experiences and instead had to piece together what happened to her based on reports from family and friends who cared for her as well as her records that were collected once she was hospitalised because she was considered a danger to herself. She was evaluated for a seizure disorder in the hospital using standard procedures, which included continuous recording of her brain electrical activity via a cap of electroencephalogram (EEG) electrodes attached to her scalp and continuous videoing of her behaviour in her room. When Susannah later watched these videos of her disorganised and bizarre behaviour and ramblings, she did not recognise herself.

She was under the care of neurologists in the hospital who were concerned she had a seizure disorder or some other neurological condition. Yet, they did not fully rule out a psychological disorder because her paranoia, hallucinations and disorganisation were so profound. And her MRIs and CT scans came back normal as did her blood tests, suggesting that she did not have an infection or a disease that had attacked the brain. The neurologists consulted psychiatrists who felt certain that she had some type of psychotic disorder. She was given antipsychotic medications. She began to develop motor symptoms that looked like catatonia. She wasn't getting any better.

Finally, the results of a second lumbar puncture (spinal tap) pointed to a possible clue. She had a very high number of white blood cells, or lymphocytes, in her cerebrospinal fluid (CSF). Lymphocytes are a part of our immune system that fights infection by releasing cytokines. Cytokines promote inflammation to help fight infection. Susannah's brain was quite literally inflamed or 'on fire' as the title of her book describes. Further neuropsychological testing (see the chapter on diagnosis and assessment) revealed that she was experiencing left neglect and this indicated that it was primarily the right side of her brain that was inflamed. Her immune system was not responding to an infection, instead it was attacking her healthy neurons as if they were infectious agents. Additional testing revealed that her immune system was attacking specific neurons with NMDA (N-methyl-D-aspartate acid) receptors. As we discuss later in the chapter, new treatments for schizophrenia are targeting NMDA receptors (if these receptors are blocked too much, psychotic symptoms can occur). Susannah was having what is called an

'autoimmune' reaction, meaning the immune system was automatically going off for no identifiable reason and it was wreaking havoc on neurons with NMDA receptors, causing the paranoia, hallucinations, catatonia and other symptoms. Her official diagnosis was 'anti-NMDA-receptor-autoimmune encephalitis', an extremely rare condition that was only first identified in 2005 (Dalmau et al., 2007; Vitaliani et al., 2005).

At the time of her diagnosis, Susannah was just the 217th person to receive the diagnosis and it took her doctors nearly a month to come to it. Cahalan poignantly wonders, 'If it took so long for one of the best hospitals in the world to get to this step, how many other people were going untreated, diagnosed with a mental illness or condemned to a life in a nursing home or a psychiatric ward?' (Cahalan, 2012, p. 151).

Given that anti-NMDA-receptor-autoimmune encephalitis is rare, it is not likely that a huge number of people with schizophrenia are misdiagnosed. In fact, recent research has found that some people early in the course of schizophrenia have the NMDA antibodies but not anti-NMDA-receptor-autoimmune encephalitis (Steiner et al., 2013). Also, a number of other antibodies have been identified as the cause of an autoimmune encephalitis including antibodies to voltage-gated potassium channel complexes and dopamine receptors (Deakin, Lennox, & Zandi, 2014; Pathmanandavel et al., 2015). Still, Cahalan presents an important cautionary tale about diagnosis of psychological disorders. Since we do not yet have a blood or brain test for schizophrenia, the diagnosis is made based on the set of observed behavioural symptoms. Yet organic causes for many emotional disorders exist and careful investigation for these other causes is important. Thus, mental health professionals would do well not to be too quick to make a diagnosis of schizophrenia or bipolar disorder or any psychological disorder, and instead consider that other factors might be contributing to the symptoms.

In Susannah's case, she was successfully treated over the course of several months with a combination of steroids to reduce the brain inflammation, plasma exchange (taking blood out of the body, treating the plasma to get rid of anti-NMDA antibodies and returning the blood) and an intravenous immunoglobulin treatment. She also attended several cognitive rehabilitation sessions to help restore cognitive functions like planning, memory and attention that were disrupted by the inflammation in her brain. She was able to return to work seven months after her hospitalisation and she wrote an article for the newspaper she worked for eight months after her diagnosis. Her book was published three years later and was released as a movie in 2016.

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Susannah Cahalan was diagnosed with anti-NMDA-receptor-autoimmune encephalitis. At the time, she was just the 217th person in the world to receive this diagnosis.



### QUESTIONS

1. Susannah's illness was caused by an inflammatory process in the brain. Is there evidence for inflammation in the brain as a cause of other psychiatric disorders?
2. Why would brain inflammation cause a hallucination?

One of the most puzzling aspects of schizophrenia is the **loss of insight** into the signs and symptoms of schizophrenia by the people who have the disorder. This can manifest as complete and terrified belief in the veracity of symptoms such as hallucinations and delusions, or a bland disagreement about the poverty-stricken and disorganised state that the person with schizophrenia might be living in. Poor insight interferes with treatment (Why would you bother cooperating with a treatment if you didn't have an illness?) and is correlated with the severity of the illness (Sevy, Nathanson, Visweswarajah, & Amador, 2004).

## Negative symptoms

The **negative symptoms** of schizophrenia consist of behavioural deficits in motivation, pleasure, social closeness and emotion expression (Kirkpatrick, Fenton, Carpenter, & Marder, 2006). These symptoms tend to endure beyond an acute episode and have profound effects on the lives of people with schizophrenia. They are also important prognostically; the presence of many negative symptoms is a strong predictor of a poor recovery (e.g., occupational impairment, few friends) two years following hospitalisation (Ho, Nopoulos, Flaum, Arndt, & Andreasen, 1998; Milev, Ho, Arndt, & Andreasen, 2005; Siegel et al., 2006). They are considered by many as the core features of the disorder.

### Avolition

Apathy, or **avolition**, refers to a lack of motivation and a seeming absence of interest in or an inability to persist in what are usually routine activities, including work or school, hobbies or social activities. For example, people with avolition may not be motivated to watch TV or contact friends. They may have difficulty persisting at work, school or household chores and may spend much of their time sitting around doing nothing. One recent study examined the types of motivation deficits in schizophrenia by interviewing people with and without schizophrenia four times a day for seven days about their daily goals. The researchers found that people with schizophrenia were less motivated by goals about autonomy (self-expression), gaining new knowledge or skills, or praise by others compared to people without schizophrenia but were more motivated by goals that had to do with reducing boredom (Gard et al., 2014). However, people with schizophrenia were equally motivated by goals that had to do with relatedness to others and with avoiding a negative outcome (e.g., criticism). Thus, it appears that people with schizophrenia may have trouble with motivation for certain life areas, but not for others.

### Asociality

Some people with schizophrenia have severe impairments in social relationships, referred to as **asociality**. They may have few friends, poor social skills and very little interest in being with other people. They may not desire close relationships with family, friends or romantic partners. Instead, they may wish to spend much of their time alone. When around others, people with this symptom may interact only superficially and briefly, and may appear aloof or indifferent to the social interaction.

### Anhedonia

A loss of interest in or a reported lessening of the experience of pleasure is called **anhedonia**. There are two types of pleasure experiences in the anhedonia construct. The first, called **consummatory pleasure**, refers to the amount of pleasure experienced in the moment or in the presence of something pleasurable. For example, the amount of pleasure you experience as you are eating a good meal is known as consummatory pleasure. The second type of pleasure, called **anticipatory pleasure**, refers to the amount of

expected or anticipated pleasure from future events or activities. For example, the amount of pleasure you expect to receive after graduating from university is anticipatory pleasure. People with schizophrenia appear to have a deficit in anticipatory pleasure but not consummatory pleasure (Gard, Kring, Germans Gard, Horan, & Green, 2007; Kring, 1999; Kring & Caponigro, 2010). That is, when asked about expected future situations or activities that are pleasurable for most people (e.g., good food, recreational activities, social interactions) on an anhedonia questionnaire, people with schizophrenia report that they derive less pleasure from these sorts of activities than do people without schizophrenia (Gard et al., 2007; Horan, Kring, & Blanchard, 2006). However, when presented with actual pleasant activities, such as amusing films or tasty beverages, people with schizophrenia report experiencing as much pleasure as do people without schizophrenia (Gard et al., 2007). Thus, the anhedonia deficit in schizophrenia appears to be in anticipating pleasure, not experiencing pleasure in the moment or in the presence of pleasurable things.

### Blunted affect

**Blunted affect** refers to a lack of outward expression of emotion. A person with this symptom may stare vacantly, the muscles of the face motionless, the eyes lifeless. When spoken to, the person may answer in a flat and toneless voice and not look at his or her conversational partner. Blunted affect was found in 66 percent of a large sample of people with schizophrenia (Sartorius et al., 1974).

The concept of blunted affect refers only to the outward expression of emotion, not to the patient's inner experience. Over 20 different studies have shown that people with schizophrenia are much less facially expressive than are people without schizophrenia; this is true in daily life or in laboratory studies when emotionally evocative stimuli (films, pictures, foods) are presented. However, people with schizophrenia report experiencing the same amount or *even more* emotion than people without schizophrenia (Kring & Moran, 2008).

### Alogia

**Alogia** refers to a significant reduction in the amount of speech. Simply put, people with this symptom do not talk much. A person may answer a question with one or two words and will not be likely to elaborate on an answer with additional detail. For example, if you ask a person with alogia to describe a happy life experience, the person might respond 'getting married' and then fail to elaborate even when asked for additional information. Along with this poverty of speech there can also be a poverty of content of thought in that ideas are simple, unelaborated and expressed with a minimum of words.

Although we have just described five different negative symptoms, research suggests that these symptoms can be understood more simply as representing two domains (Blanchard & Cohen, 2006; Kring, Gur, Blanchard, Horan, & Reise, 2013; Messinger et al., 2011). The first domain, involving motivation, emotional experience and sociality, is sometimes referred to as the *motivation and pleasure* domain. The second domain, involving outward expression of emotion and vocalisation, is referred to as the *expression* domain.

## Disorganised symptoms

**Disorganised symptoms** include disorganised speech and disorganised behaviour.

### Disorganised speech

Also known as *formal thought disorder*, **disorganised speech** refers to problems in organising ideas and in speaking so that a listener can understand. The following excerpt illustrates the incoherence sometimes found in the conversation of people with schizophrenia as an interviewer tries to ask John, a person with schizophrenia, several questions.

**Interviewer:** Have you been nervous or tense lately?

**John:** No, I got a head of lettuce.

**Interviewer:** You got a head of lettuce? I don't understand.

**John:** Well, it's just a head of lettuce.

**Interviewer:** Tell me about lettuce. What do you mean?

**John:** Well... lettuce is a transformation of a dead cougar that suffered a relapse on the lion's toe. And he swallowed the lion and something happened. The... see, the... Gloria and Tommy, they're two heads and they're not whales. But they escaped with herds of vomit and things like that.

**Interviewer:** Who are Tommy and Gloria?

**John:** Uh,... there's Joe DiMaggio, Tommy Henrich, Bill Dickey, Phil Rizzuto, John Esclavera, Del Crandell, Ted Williams, Mickey Mantle, Roy Mande, Ray Mantle, Bob Chance...

**Interviewer:** Who are they? Who are those people?

**John:** Dead people... they want to be f\*\*\*ed... by this outlaw.

**Interviewer:** What does all that mean?

**John:** Well, you see, I have to leave the hospital. I'm supposed to have an operation on my legs, you know. And it comes to be pretty sickly that I don't want to keep my legs. That's why I wish I could have an operation.

**Interviewer:** You want to have your legs taken off?

**John:** It's possible, you know.

**Interviewer:** Why would you want to do that?

**John:** I didn't have any legs to begin with. So I would imagine that if I was a fast runner, I'd be scared to be a wife, because I had a splinter inside of my head of lettuce. (Neale & Oltmanns, 1980, pp. 103–104)

Although John may make repeated references to central ideas or themes, the images and fragments of thought are not connected; it is difficult to understand what he is trying to tell the interviewer.

Speech may also be disorganised by what are called **loose associations**, in which ideas sound disjointed — they slip into each other, though are still related, and the person appears to have difficulty sticking to one topic. As the logical order of thought is further degraded, thoughts are juxtaposed without a meaningful relationship. This is also termed **derailment**.

**Interviewer:** What did you think of the scandal?

**Peter:** You know I didn't tune in on that, I felt so bad about it. I said, boy, I'm not going to know what's going on in this. But it seemed to get so murky and everybody's reports were so negative. Huh, I thought, I don't want any part of this and I was I don't care who was in on it and all I could figure out was he had something to do with it. He was trying to flush the bathroom toilet or something. She was trying to do something fairly simple. The tour guests stuck or something. She got blamed because of the water overflowed, went down in the basement, down, to the kitchen. They had a, they were going to have to repaint and restore the enormous living room. And then it was at this reunion they were having. And it's just such a mess and I just thought, well, I'm just going to pretend like I don't even know what's going on. So I came downstairs and 'cause I pretended like I didn't know what was going on, I slipped on the floor of the kitchen, cracking my toe, when I was teaching some kids how to do some double dives. (Adapted from Andreasen, 1979, p. 1319).

As the degree of thought disorder worsens, the person's speech becomes unintelligible as grammar and content are broken down into smaller sections of seemingly unrelated words. This is known as *incoherence*.

**Interviewer:** Why do you think people believe in God?

**Amber:** Urn, because making a do in life. Isn't none of that stuff about evolution guiding isn't true anymore now. It all happened a long time ago. It happened in eons and eons and stuff they wouldn't believe in him. The time that Jesus Christ people believe in their thing people believed in, Jehovah God that they didn't believe in Jesus Christ that much. (Andreasen, 1979, p. 1319)

At its worst, disorganised speech is described as *word salad*. Speech resembles a Dada poem, with words or even syllables chopped up and interspersed in a totally unintelligible way.

People with schizophrenia have an awareness of this problem and have described what it is like to experience disorganised speech.

My thoughts get all jumbled up. I start thinking or talking about something but I never get there. Instead, I wander off in the wrong direction and get caught up with all sorts of different things that may be connected with things I want to say but in a way I can't explain. People listening to me get more lost than I do. My trouble is that I've got too many thoughts. You might think about something, let's say that ashtray and just think, oh yes, that's for putting my cigarette in, but I would think of it and then I would think of a dozen different things connected with it at the same time. (Quoted in McGhie & Chapman, 1961, p. 108)

It would seem logical to expect disorganised speech to be associated with problems in language production, but this does not appear to be the case. Instead, disorganised speech is associated with problems in what is called executive functioning — problem solving, planning and making associations between thinking and feeling. Disorganised speech is also related to the ability to perceive semantic information (i.e., the meaning of words) (Kerns & Berenbaum, 2002, 2003).

### Disorganised behaviour

People with the symptom of **disorganised behaviour** may go into inexplicable bouts of agitation, dress in unusual clothes, act in a silly manner, hoard food or collect garbage. They seem to lose the ability to organise their behaviour and make it conform to community standards. They also have difficulty performing the tasks of everyday living.

In DSM-5, one manifestation of disorganised behaviour is called **catatonia**. People with this symptom may gesture repeatedly, using peculiar and sometimes complex sequences of finger, hand and arm movements, which often seem to be purposeful. Some people manifest an unusual increase in their overall level of activity, including much excitement, flailing of the limbs and great expenditure of energy similar to that seen in mania. At the other end of the spectrum is immobility: people adopt unusual postures and maintain them for very long periods of time. Catatonia can also involve waxy flexibility — another person can move the person's limbs into positions that the person will then maintain for long periods of time.

Catatonia is seldom seen today in people with schizophrenia in developed countries, perhaps because medications work effectively on these disturbed movements or postures. While once a subtype of schizophrenia, catatonia is now thought of as a neuropsychiatric syndrome that can be seen in mood disorders, neurological diseases, drug intoxication and developmental disorders as well as in schizophrenia (Weder, Muralee, Penland, & Tampi, 2008). See focus on discovery 6.2 for more details on the history of schizophrenia and its symptoms.

### Cognition

Schizophrenia is not only a disorder of positive and negative symptoms, it is a disorder of cognition. Cognitive deficits in schizophrenia are common and include both neurocognition (attention, concentration, memory, speed of processing and executive functioning) and social cognition (emotion recognition, theory of mind, attributional bias) (Heinrichs & Zakzanis, 1998; Mesholam-Gately, Giuliano, Goff, Faraone, & Seidman, 2009). Although these difficulties are not as easily assessed as hallucinations and delusions, they are even more significant than the positive symptoms in determining functional outcome (Green, Kern, & Heaton, 2004). These deficits are demonstrable prior to the onset of schizophrenia (Woodberry, Giuliano, & Seidman, 2008), worsen at the time of first presentation of schizophrenia and then appear to stabilise (Mesholam-Gately et al., 2009). These deficits appear to be broad rather than selective and vary in degree from mild to very severe. This leaves individuals with schizophrenia still capable of working, studying and playing their part in our communities; however, it does limit their potential.

It is not hard to see how cognitive deficits interfere with day-to-day functioning. Problems with attention and concentration make it difficult to register new information and to follow events. Memory deficits make it harder to retain information in the workplace or at school. The decreased speed of processing may make it harder to keep up with others at work. Social cognitive deficits are a major barrier for many

(Couture, Penn, & Roberts, 2006; Fett, Maat, & Investigators, 2011) undermining the ability of individuals with schizophrenia to recognise the emotional state of people, leaving them alienated from the interaction between colleagues and prone to distortions in their explanatory model for what is happening around them (Couture et al., 2006).

## Anxiety and depression

Anxiety and depression are very common in people with schizophrenia, both as part of the syndrome of the disorder and as a comorbid condition such as panic disorder or major depressive disorder (Buckley, Miller, Lehrer, & Castle, 2009). These symptoms significantly contribute to the level of disability and mortality, with approximately 5 percent of people with schizophrenia ending their own lives by suicide (Palmer et al., 2005).

The interplay of symptoms and stages of recovery is complex. Anxiety and depressive symptoms are commonly observed as part of the prodrome of schizophrenia — the period during the development of the disorder when the individual may not demonstrate all of the features needed for diagnosis. Prospectively, this has become known as the *at-risk mental state* (Yung et al., 2005). The range of symptoms seen is wide with depression, demoralisation (Restifo, Harkavy-Friedman, & Shrout, 2009), panic attacks, obsessions, compulsions, rituals and social anxiety all observed. During the period of recovery from the initial acute stages of schizophrenia, the interplay between symptoms can be ambiguous; for example, differentiating between residual persecutory fears and social avoidance as part of social anxiety can be difficult.

The recovery of people from schizophrenia can be a rocky road with periods of dejection, demoralisation and despair (Drake, Whitaker, Gates, & Cotton, 1985). This is frequently worse early in the course of recovery, with responses to the diagnosis of schizophrenia including withdrawal, substance abuse and suicide (Addington, Addington, & Patten, 1998). Although this may be conceptualised as a separate disorder to schizophrenia, the identification and treatment of depressive symptoms is very important to the quality of life of the person with schizophrenia (Meijer, Koeter, Sprangers, & Schene, 2009; Wegener et al., 2005).

### FOCUS ON DISCOVERY 6.2

#### History of the concept of schizophrenia

Two European psychiatrists, Emil Kraepelin and Eugen Bleuler, initially formulated the concept of schizophrenia. Kraepelin first described **dementia praecox**, his term for what we now call schizophrenia, in 1898. Dementia praecox included several diagnostic subtypes — dementia paranoides, catatonia and hebephrenia — that had been regarded as distinct entities by clinicians in the previous few decades. Although these disorders were symptomatically diverse, Kraepelin believed that they shared a common core. The term *dementia praecox* reflected what he believed was that core — an early-onset (praecox) and a progressive, inevitable intellectual deterioration (dementia). The dementia in dementia praecox is not the same as the dementias we discuss in the chapter on neurocognitive disorders, which are defined principally by severe memory impairments. Kraepelin's term referred to a general 'mental enfeeblement'.

Bleuler broke with Kraepelin's description on two major points: he believed that the disorder did not necessarily have an early onset and he believed that it did not inevitably progress towards dementia. Thus the label 'dementia praecox' was no longer appropriate and in 1908 Bleuler proposed his own term, *schizophrenia*, from the Greek words *schizein* ('to split') and *phren* ('mind'), capturing what he viewed as the essential nature of the condition. He concentrated on what would become known as Bleuler's 4 'A' — loosening of associations, disturbance of affect, autism and ambivalence.

Emil Kraepelin (1856–1926), a German psychiatrist, articulated descriptions of schizophrenia (then called dementia praecox) that have proved remarkably durable in the light of contemporary research.

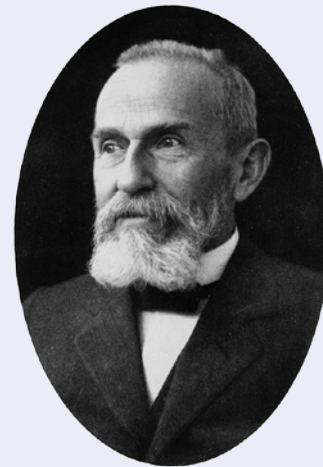


With age of onset and deteriorating course no longer considered defining features of the disorder, Bleuler faced a conceptual problem. The symptoms of schizophrenia could vary widely among people, so he had to provide some justification for putting them into a single diagnostic category. That is, he needed to specify some common denominator or essential property that would link the various disturbances. The metaphorical concept that he adopted for this purpose was the ‘breaking of associative threads’.

For Bleuler, associative threads joined not only words but also thoughts. Thus, goal-directed, efficient thinking and communication were possible only when these hypothetical structures were intact. The notion that associative threads were disrupted in people with schizophrenia could then be used to account for the range of other disturbances. Bleuler viewed attentional difficulties, for example, as resulting from a loss of purposeful direction in thought, in turn causing passive responses to objects and people in the immediate surroundings.

Kraepelin had recognised that a small percentage of people with symptoms of dementia praecox did not deteriorate, but he preferred to limit this diagnostic category to people who had a poor prognosis. Bleuler’s work, in contrast, led to a broader concept of the disorder. He diagnosed some people with a good prognosis as having schizophrenia and he also diagnosed schizophrenia in many people who would have received different diagnoses from other clinicians. These different concepts of schizophrenia would cause difficulty later with North American psychiatrists taking up a very broad Bleulerian concept of schizophrenia, unlike most of the rest of the world. This led to people with bipolar disorder and borderline personality disorder being diagnosed with schizophrenia. Recognition that this was a problem would in turn lead to substantial reform of the DSM diagnostic system, bringing it in line with international diagnostic practices.

Eugen Bleuler (1857–1939), a Swiss psychiatrist, contributed to our conceptions of schizophrenia and coined the term.



#### QUESTIONS

1. Is schizophrenia a neurodegenerative or dementing illness?
2. Are there problems with using a broad, inclusive diagnostic label such as schizophrenia?

## Schizophrenia and the DSM-5

The DSM-5 requires that symptoms last at least six months for the diagnosis of schizophrenia to be made. The six-month period must include at least one month of an acute episode or active phase, defined by the presence of at least two of the following symptoms: delusions, hallucinations, disorganised speech, disorganised behaviour and negative symptoms, one of which has to be delusions, hallucinations or disorganisation. The remaining time required for the diagnosis can occur either before or after the active phase. This time criterion eliminates people who have a brief psychotic episode and then recover quickly.

The traditional subtypes of schizophrenia (i.e., paranoid, disorganised, catatonic, undifferentiated) are no longer used in the classification as they were of questionable usefulness, poor reliability and poor predictive validity (Braff, Ryan, Rissling, & Carpenter, 2013).

The differential diagnosis of schizophrenia as indicated in Susannah’s story in focus on discovery 6.1 includes possible neurological and metabolic causes of psychosis. Two other psychotic disorders that should be considered are **schizophreniform disorder** and **brief psychotic disorder**. The symptoms of schizophreniform disorder are the same as those of schizophrenia but last only one to six months. Brief psychotic disorder lasts from one day to one month and can be brought on by extreme stress. DSM-5 made only one change regarding these two disorders: the symptoms must include hallucinations, delusions or disorganised speech. **Schizoaffective disorder** comprises a mixture of symptoms of schizophrenia and mood disorders. The DSM-5 requires either a depressive or manic episode rather than simply mood disorder symptoms, as in DSM-IV-TR.

A person with **delusional disorder** is troubled by persistent delusions. These can be delusions of persecution or jealousy, such as the unfounded conviction that a spouse or lover is unfaithful. Other delusions seen in this disorder include grandiose delusions, delusions of erotomania (believing that one is loved by some other person, usually a complete stranger with a higher social status) and somatic delusions (e.g., delusions about body functions).

Other conditions that can present in a similar fashion to schizophrenia include the manic phase of a bipolar disorder when people can appear elevated in mood, highly disorganised and psychotic. Psychotic symptoms can also occur in the context of substance abuse (intoxication or withdrawal) of many psychoactive substances. The DSM-5 added a new category to the ‘Conditions for further study’ part of Section III, called *attenuated psychosis syndrome*. We discuss this disorder in more detail in focus on discovery 6.3.

### FOCUS ON DISCOVERY 6.3

#### Attenuated psychosis syndrome

The idea for attenuated psychosis syndrome (APS) came from research over the past two decades that has sought to identify young people who are at risk for developing schizophrenia. These types of studies are called **clinical high-risk studies**. The starting point for these prospective, longitudinal studies is the identification of youth who are at elevated risk of developing psychosis, perhaps because of increased genetic risk — because, for example, their mother has schizophrenia (Mednick, Cudeck, Griffith, Talovic, & Schlusinger, 1984) — or because they present with mild positive symptoms that might later develop into schizophrenia. This latter approach was adopted at the PACE clinic in Melbourne (Yung & McGorry, 1996) who identified three groups of young people at high risk of developing schizophrenia: those who have attenuated symptoms, those who have full-blown psychotic symptoms but for a brief period of time, or those who suffer a marked deterioration in their level of functioning in the context of a strong family history of psychosis. Young people who meet criteria for one of these groups differ from young people who do not meet these criteria in a number of domains, including their everyday functioning and their rate of transition to a psychotic disorder such as schizophrenia (Fusar-Poli et al., 2012). Risk increased with time, with 22 percent transitioning to psychosis after one year and 36 percent transitioning after three years (Fusar-Poli et al., 2012) compared with only 0.2 percent of the general population (Carpenter & van Os, 2011; Yung et al., 2012).

The enthusiasm for early identification of severe mental illness reflects the recognition that many people have experienced psychotic symptoms for prolonged periods of time before coming to care. A longer duration of undiagnosed psychosis (DUP) is associated with a higher symptom load, poorer overall functioning and a reduced likelihood of gaining remission (Marshall et al., 2005). Also, even if the person with APS did not go on to develop schizophrenia, they were at high risk of developing a persistent non-psychotic mental disorder (Lin et al., 2015), so identifying people with APS and bringing them to care is important.

However, there have been a number of arguments against recognising APS as a new diagnostic category (Yung et al., 2012). First, the category itself does not yet have enough reliability and validity to support its use as a formal diagnosis. Second, there is a high level of comorbidity with prodromal (early) symptoms: over 60 percent of young people meeting the prodromal criteria have a history of depression, raising questions as to where in the present classification APS should be: is it part of a mood disorder or a schizophrenia spectrum disorder? Third, there is concern that applying a new diagnostic label, particularly to young people, might be stigmatising or lead to discrimination. Because not all people with APS will develop schizophrenia, it may unnecessarily alarm young people and their families. Finally, while providing treatment for people with distressing or disabling attenuated positive symptoms is a laudable goal, there is concern that the treatment will too closely resemble that for schizophrenia, further blurring the line between the two conditions. Indeed, there is not yet a clear consensus on treatment for APS (Carpenter & van Os, 2011).

#### QUESTIONS

1. Why is it important to identify people early in the course of a severe mental illness?
2. How might stigma affect the life of a person identified with APS?

## 6.2 Aetiology of schizophrenia

**LEARNING OUTCOME 6.2** Differentiate the genetic factors, both behavioural and molecular, in the aetiology of schizophrenia.

What can explain the scattering and disconnection of thoughts, delusions, hallucinations, amotivation, diminished emotion expression and cognitive deficits of people with schizophrenia? As we will see, a number of factors contribute to the cause of this complex disorder.

### Genetic factors

A good deal of research supports the idea that schizophrenia has a genetic component, as we discuss in the following sections on behaviour genetics and molecular genetics research. The evidence is somewhat more convincing from behaviour genetics studies, largely because they have been well replicated. Current evidence indicates that schizophrenia is genetically heterogeneous — that is, genetic factors may vary from case to case — mirroring the fact, noted earlier, that schizophrenia is symptomatically heterogeneous. As is the case for any gene or genes, the genes associated with schizophrenia do their work via the environment, so gene–environment interaction studies are likely to help pinpoint the nature of the genetic contribution to schizophrenia (Walker, Mittal, & Tessner, 2008).

### Behaviour genetics research

Family, twin and adoption studies support the idea that genetic factors play a role in schizophrenia. Many behaviour genetic studies of schizophrenia were conducted when the definition of schizophrenia was considerably broader than it is now. However, behaviour genetics investigators collected extensive descriptive data on their samples, allowing the results to be reanalysed later using newer diagnostic criteria.

#### Family studies

Family studies are a well-established methodology that has consistently informed us over 40 years that genetic risk is the main driver for the development of schizophrenia. Table 6.2 clearly shows that risk follows a gradient from the person with whom an individual with schizophrenia (the proband) would share all of their genetic material (a monozygotic twin) to far more distal family members such as grandchildren. Other studies have found that people with schizophrenia in their family histories have more negative symptoms than those whose families are free of schizophrenia (Malaspina, Goetz, Yale et al., 2000); this finding suggests that negative symptoms may have a stronger genetic component.

**TABLE 6.2** Summary of major family and twin studies of the genetics of schizophrenia

Relation to proband	Percentage with schizophrenia
Spouse	1.00
Grandchildren	2.84
Nieces/nephews	2.65
Children	9.35
Siblings	7.30
DZ twins	12.08
MZ twins	44.30

*Source:* Gottesman, McGuffin, & Farmer (1987).

An important issue is that schizophrenia shares its inheritance with a range of other mental disorders. Children of parents with schizophrenia are at an increased risk of schizophrenia but also bipolar disorder and other psychiatric illnesses (Rasic, Hajek, Alda, & Uher, 2014). As you might expect, the incidence of schizophrenia is highest for children who have parents with schizophrenia. However, just having a parent with a severe mental illness will increase the risk of developing schizophrenia. These findings suggest that there may be some shared genetic vulnerability between schizophrenia and other severe mental illnesses like bipolar disorder; molecular genetics studies, which we turn to shortly, also suggest this link.

Family studies show that genes likely play a role in schizophrenia, but of course the relatives of a person with schizophrenia share not only genes but also common experiences. It has been estimated that while the genetic liability for schizophrenia accounted for 81 percent of the risk, there is an effect for the shared environment of 11 percent for twins. That is, although most of the risk of developing schizophrenia in monozygotic or dizygotic twins was coded in the genes, a significant effect (11 percent) was caused by the environment that the twin grew up in (Sullivan, Kendler, & Neale, 2003). Therefore, the influence of the environment cannot be discounted in explaining the higher risks among relatives.

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Behaviour genetics studies often focus on twins or, more rarely, triplets and quadruplets. In one rare case, all of the Genain quadruplet girls (not pictured), born in 1930, developed schizophrenia. Two of the still surviving sisters were recently evaluated at age 81 and both were still taking antipsychotic medication (Mirsky, Bieliauskas, Duncan, & French, 2013).



### **Twin studies**

Table 6.2 also shows the risk for identical (MZ) and fraternal (DZ) twins of people with schizophrenia. The risk for MZ twins (44.3 percent), though greater than that for DZ twins (12.08 percent), is still much less than 100 percent. Similar results have been obtained in more recent studies (Cannon, Kaprio, Lonnqvist, Huttunen, & Koskenvuo, 1998; Cardno, Marshall, Coid et al., 1999). The less-than-100-percent concordance in MZ twins is important: if genetic transmission alone accounted for schizophrenia and one identical twin had schizophrenia, the other twin would also have schizophrenia. Twin study research also suggests that negative symptoms may have a stronger genetic component than do positive symptoms (Dworkin, Lenzenwenger, & Moldin, 1987; Dworkin & Lenzenwenger, 1984).

As with family studies, of course, there is a critical problem in interpreting the results of twin studies. Common shared (e.g., child-rearing practices) and non-shared (e.g., peer relationships) environmental factors rather than common genetic factors could account for some portion of the increased risk.

A clever analysis supporting a genetic interpretation of the high risk found for identical twins was performed by Fischer (1971). She reasoned that if these rates indeed reflected a genetic effect, the twins without schizophrenia would presumably carry risk genes for schizophrenia, even though it was not expressed behaviourally and thus might pass along an increased risk for the disorder to their children. Indeed, the rate of schizophrenia and schizophrenia-like psychoses in the children of the MZ twins without schizophrenia was 9.4 percent, while the rate among the children of the twins with schizophrenia

was only slightly and non-significantly higher, 12.3 percent. Both rates are substantially higher than the 1 percent prevalence found in the general population, which lends further support to the importance of genetic factors in schizophrenia.

### Adoption studies

The study of children whose biological mothers had schizophrenia but who were reared from early infancy by adoptive parents without schizophrenia is another useful behaviour genetics study method. Such studies eliminate the possible effects of being reared in an environment where a parent has schizophrenia. A fivefold increase in the odds ratio for developing schizophrenia is seen in the offspring of mothers with schizophrenia who place their child for adoption compared to a control population (Sullivan, 2005).

**TABLE 6.3 Summary of family, adoption and twin studies in schizophrenia**

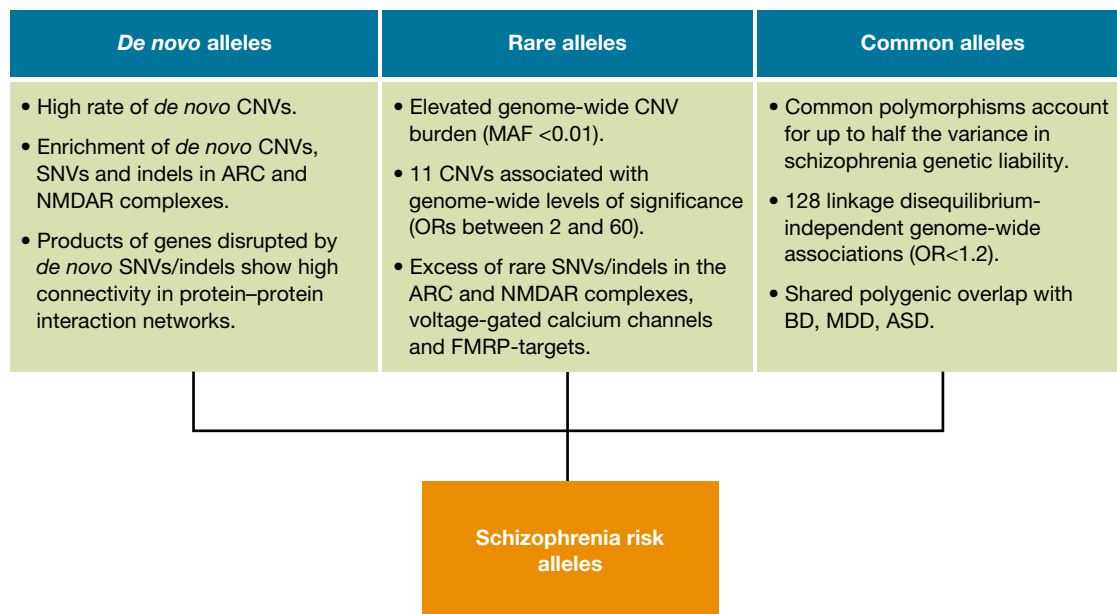
Study type	Conceptual basis	Studies	Findings
Family	Risk of schizophrenia in first-degree relatives of cases with schizophrenia vs. controls	11	10/11 studies show familiarity of schizophrenia Significant familial aggregation of schizophrenia; odds ratio: 9.8 (95% CI 6.2–15.5)
Adoption	Risk of schizophrenia in adoption cluster (offspring of one set of parents raised from early in life by unrelated strangers)	5	Effect of postnatal environment negligible Adoptees with schizophrenia: increased risk in biological vs. adoptive parents (OR = 5.0; 95% CI 2.4–10.4) Parents with schizophrenia: increased risk in biological vs. control offspring 3.5 (95% CI 1.9–6.4)
Twin	Risk of schizophrenia in monozygotic vs. dizygotic twins	12	Heritability in liability to schizophrenia: 81% (95% CI 73–90%) Environmental effects shared by members of a twin pair: 11% (95% CI 3–19%)

*Source:* Sullivan (2005).

### Molecular genetics research

Knowing that schizophrenia has a genetic component is in many ways just the starting point for research. Understanding exactly what constitutes the genetic predisposition is the challenge faced by molecular genetics researchers. As with nearly all of the disorders we cover in this text, the predisposition for schizophrenia is not transmitted by a single gene. Furthermore, recent molecular genetics research has found that there are multiple common genes associated with schizophrenia, bipolar disorder, major depression and autistic spectrum disorder, the strongest being the link between schizophrenia and bipolar disorder (Bulik-Sullivan et al., 2015). We are beginning to understand how the genes that confer risk for schizophrenia are related to underlying genotypic networks that are active in interconnected pathways governing neural transmission, neural development and function (Arnedo et al., 2015). These pathways do not relate directly to clinical symptoms and are shared across multiple disorders, which explains the difficulty that we have had in clearly delineating the phenotype of schizophrenia from disorders such as bipolar disorder.

The genetic risks for schizophrenia can be understood as deriving from one of three general classes: (1) copy number variations (CNV) that have arisen *de novo*, (2) rare alleles or CNV of high risk and (3) common polymorphisms of low risk. Recall from chapter 1 that genetic research allows researchers to identify rare mutations, such as CNVs (copy number variations), in genes rather than just known gene loci. Mutations are changes in a gene that occur randomly and for unknown reasons. A CNV refers to an abnormal copy (a deletion or a duplication) of one or more sections of DNA in a gene (see chapter 1).

**FIGURE 6.1** Schizophrenia risk alleles

Source: Rees, O'Donovan, & Owen (2015).

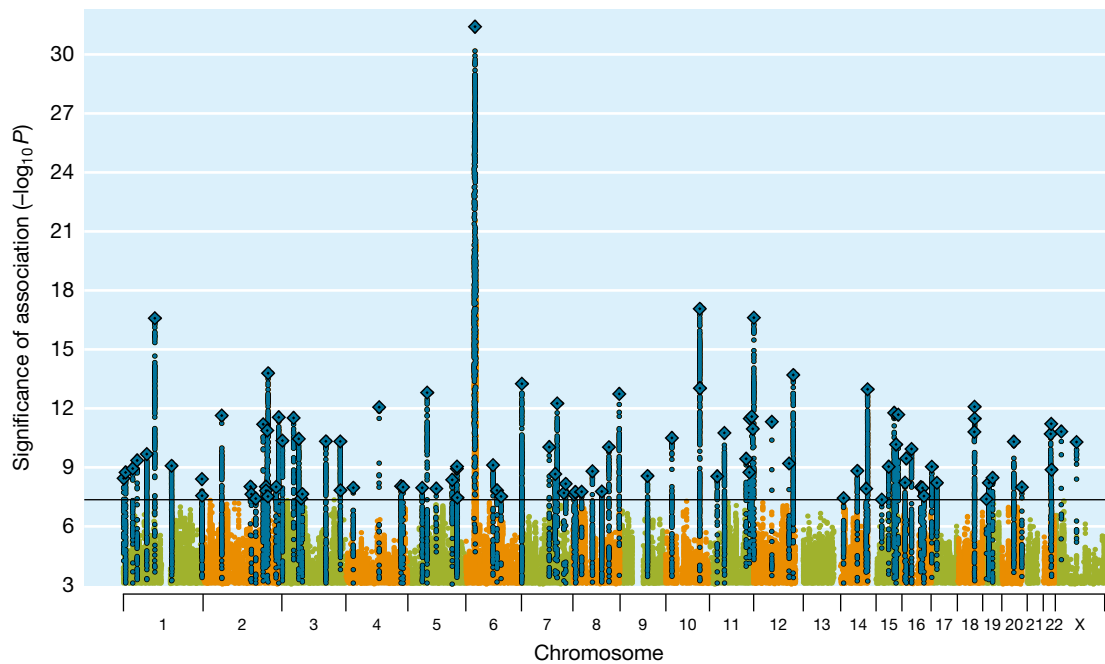
*De novo* risk alleles refer to the creation of new mutations in the genetic code. This rare though potent cause of disease is found at an increased rate in schizophrenia (approximately 5 percent) as compared to controls (2%) (Rees, O'Donovan, & Owen, 2015), as well as in a range of other neurodevelopmental disorders such as autism and mental retardation. It is associated with increasing paternal age (McGrath et al., 2014). In the case of individuals who go on to develop schizophrenia, these new genetic materials frequently are associated with the glutamatergic synapse involving both activity-regulated cytoskeleton-associated protein (ARC) and N-methyl-D-aspartate receptor (NMDAR) complexes, as well as proteins that regulate synaptic strength. These mutations overlapped with those found in autism and other neurodevelopmental disorders (Fromer et al., 2014).

A small number of rare inherited CNV abnormalities have also been associated with schizophrenia. There is frequently an overlap with other neurodevelopmental disorders such as velocardiofacial syndrome, autism, ADHD and Prader-Willi syndrome that share the CNV abnormalities and some of the phenotypic expression (Rees et al., 2015). These abnormalities include duplications and deletions of genetic material that are related to NMDA receptor function, metabotropic glutamate receptor and calcium channel signalling and track to these and other protein–protein pathway interactions.

Much of the genetic risk for schizophrenia is associated with common alleles of low risk. These have been detected on a large scale with the introduction of genome-wide association studies (GWAS). An important advance was the publication by the Psychiatric Genomics Consortium of a GWAS of 36 989 people with schizophrenia. This study greatly expanded the number of genes known to be associated with schizophrenia to 128 at 108 loci (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014). The study emphasised the polygenic nature of schizophrenia, giving support to the conception of schizophrenia as a syndrome with multiple overlapping possible aetiologies. Genes associated with the dopamine D2 receptor (*DRD2*), glutamatergic transmission (*GRM3*, *GRIN2A*, *SRR*, *GRIAI*), calcium channel proteins and synaptic proteins were consistent with prior findings, and with proposed mechanisms of aetiology or treatment. In addition, the study pointed to a possible strong role for immune dysfunction in schizophrenia. In fact, the strongest association identified was with the genes that coded for the major histocompatibility complex (see figure 6.2). This insight has already

provided researchers with a mechanism for the dysfunction in synaptic pruning that is known to occur in schizophrenia. In a groundbreaking series of experiments, Sekar and colleagues (Sekar et al., 2016) identified a number of variants of complement coded at the MHC spike associated with schizophrenia. A specific variant of the complement *C4* gene was found to be increased in people with schizophrenia and expressed at a higher rate in the brains of people with schizophrenia. This variant was associated with an increase in the elimination of synapses due to an interaction with microglia in the brain. This has been heralded as the first convincing physiological explanation for brain changes in schizophrenia (Dhindsa & Goldstein, 2016).

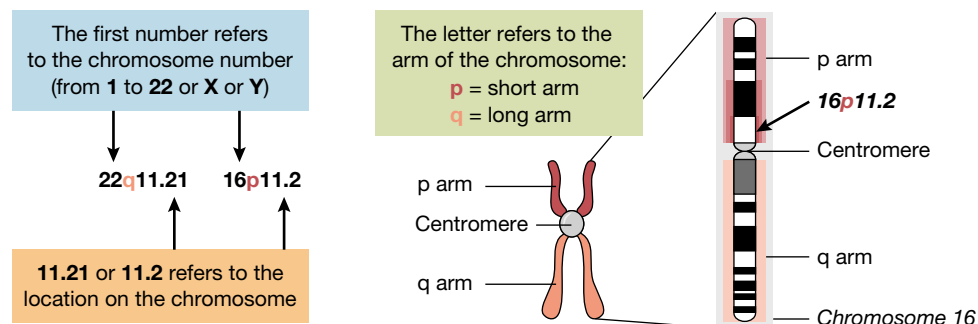
**FIGURE 6.2** Manhattan plot showing 128 genes of interest in schizophrenia



**Source:** Schizophrenia Working Group of the Psychiatric Genomics Consortium (2014).

**FIGURE 6.3** Decoding the language of genes

**What do all the letters and numbers mean?**



## 6.3 The role of neurotransmitters

**LEARNING OUTCOME 6.3** Describe how the brain has been implicated in schizophrenia.

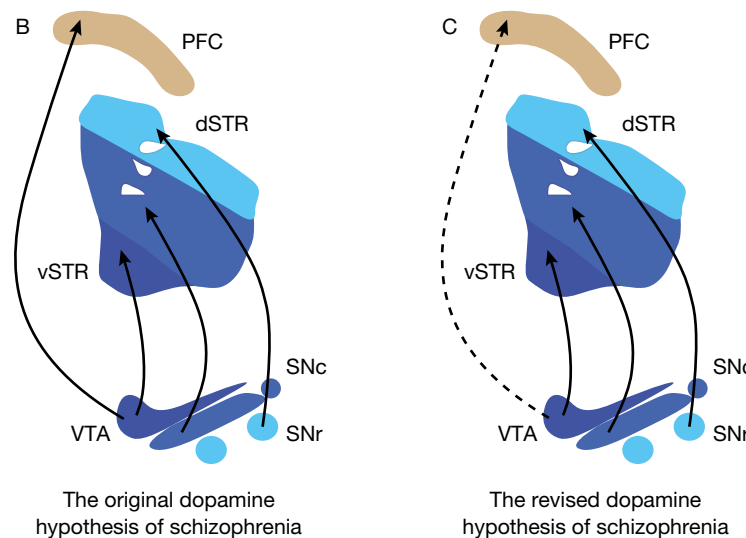
Present research is examining several different neurotransmitters, such as serotonin and glutamate, to see what role they might play in the aetiology of schizophrenia. The first neurotransmitter to receive substantial research attention was dopamine. This research has both helped and hindered efforts to identify causes and treatments for schizophrenia.

### Dopamine theory

The theory that schizophrenia is related to excess activity of the neurotransmitter dopamine is based principally on the knowledge that drugs effective in treating schizophrenia reduce dopamine activity. Researchers have noted that antipsychotic drugs, in addition to being useful in treating some symptoms of schizophrenia, produce adverse effects resembling the symptoms of Parkinson's disease. Parkinson's disease is caused in part by low levels of dopamine in a particular area of the brain. Antipsychotic drugs fit into and thereby block a particular type of postsynaptic dopamine receptor, called D2 receptors. Further indirect support for the dopamine theory of schizophrenia came from findings that antipsychotics were able to block the psychomotor activity associated with stimulant medications such as amphetamines (Baumeister & Francis, 2002) and the recognition that stimulant medications could produce a state that closely resembles schizophrenia in people who do not have the disorder; they can also exacerbate the symptoms of people with schizophrenia (Angrist, Lee, & Gershon, 1974).

**FIGURE 6.4**

(B) 'The original dopamine hypothesis of schizophrenia. The original dopamine hypothesis proposed that a global hyperactivity of the dopaminergic projections in the brain may lead to the symptoms of schizophrenia. (C) The revised dopamine hypothesis of schizophrenia. The revised dopamine hypothesis proposed that a hyperactive nigrostriatal dopaminergic projection leads to positive symptoms but a hypoactive mesocortical projection is responsible for cognitive and negative symptoms' (Simpson, Kellendonk, & Kandel, 2010, p. 587).



Source: Simpson et al. (2010).

A recent meta-analysis of work with SPECT and PET scans targeting dopamine receptor and transporter changes in schizophrenia has concluded that excess striatal dopamine drives the positive and disorganisation symptoms of schizophrenia and that this excess is most probably presynaptic (Howes et al., 2012). It is not accompanied by significant changes in post-synaptic D2 receptors or changes in dopamine

transporter function. Antipsychotic medications suppress these symptoms by blocking dopamine D2 receptors in the mesolimbic pathway, thereby lowering dopamine activity; however, they do not alter the underlying pathology that is driving the symptomatology. Hence any non-adherence or hasty reduction in medication dose may result in a rapid return of symptoms. These changes in the striatum are accompanied by a decreased dopamine release in the **prefrontal cortex**, which has been related to both the negative and cognitive symptoms of schizophrenia (Slifstein et al., 2015).

Because schizophrenia is a disorder with widespread symptoms related to perception, emotion, cognition and social behaviour, it is unlikely that a single neurotransmitter could account for all symptoms. Thus, schizophrenia researchers have cast a broader neurotransmitter net, moving away from an emphasis on dopamine.

### Other neurotransmitters

As we discuss later, newer drugs used in treating schizophrenia implicate other neurotransmitters, such as serotonin, in the disorder. These newer drugs partially block D2 receptors, but they also work by blocking the serotonin receptor 5-HT<sub>2A</sub> (Burris et al., 2002). Dopamine neurons generally modulate the activity of other neural systems; for example, in the prefrontal cortex they regulate gamma-aminobutyric acid (GABA) neurons. Thus, it is not surprising that GABA transmission is disrupted in the prefrontal cortex of people with schizophrenia (Volk, Austin, Pierri et al., 2000). Similarly, serotonin neurons regulate dopamine neurons in the mesolimbic pathway.

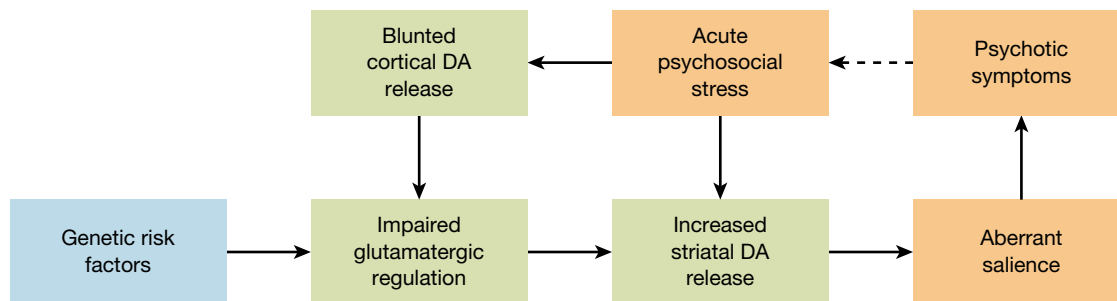
Glutamate, a neurotransmitter that is widespread in the human brain, may also play a role (Carlsson, Hanson, Waters, & Carlsson, 1999). Low levels of glutamate have been found in the cerebrospinal fluid of people with schizophrenia (Faustman, Bardgett, Faull, Pfefferman, & Sceransky, 1999) and postmortem studies have revealed low levels of the enzyme needed to produce glutamate (Tsai et al., 1995). Studies have found elevated levels of the amino acid homocysteine, a substance that is known to interact with the NMDA receptor among people with schizophrenia and, during their third trimester, in the blood of pregnant women whose offspring developed schizophrenia as adults (Brown et al., 2007; Regland, Johansson, Grenfeldt, Hjelmgren, & Medhus, 1995). The anaesthetic phencyclidine (PCP) can induce both positive and negative symptoms in people without schizophrenia by antagonising glutamate at NMDA receptors (Gouzoulis-Mayfrank et al., 2005; O'Donnell & Grace, 1998). Furthermore, a decrease in glutamate inputs from either the prefrontal cortex or the hippocampus (both of these brain structures are implicated in schizophrenia) to the corpus striatum could result in increased dopamine activity (O'Donnell & Grace, 1998). Additional evidence suggests that cognitive deficits in schizophrenia supported by the prefrontal cortex as well as symptoms of disorganisation may be connected to deficits involving NMDA (MacDonald & Chafee, 2006).

Can these perspectives be integrated? Genetic work points to the involvement of dopaminergic, glutamatergic and GABAergic systems. These changes clearly increase the vulnerability of the brain to aberrant neurodevelopment and the effects of stress. We suspect that this system is primed for the development of schizophrenia by dysfunction in glutamatergic (excitatory) control, leading to a loss of regulatory feedback in the striatum contributing to the increase in striatal dopamine (see figure 6.5). It is this spiking of dopaminergic activity that is associated with disorganisation and positive symptoms. The decrease in glutamatergic activity and associated dopaminergic activity in the prefrontal cortex (PFC) is accompanied by the negative and cognitive symptoms of schizophrenia. These changes are also related to alterations in the underlying cortical network, which we will review below.

## Brain structure and function

Because schizophrenia affects so many domains (thought, emotion and behaviour), it makes sense that a single region or site cannot account for all of schizophrenia's symptoms. Among the most well-replicated findings of brain abnormalities in schizophrenia are enlargement of the ventricles and dysfunction in the prefrontal cortex and temporal cortex, as well as surrounding brain regions. Newer research has identified problems in how different areas of the brain are connected to one another.

**FIGURE 6.5** Integrating neurotransmitter and genetic factors leading to psychosis



*Source:* Howes, McCutcheon, Owen, & Murray (2017).

### Enlarged ventricles

Postmortem studies of the brains of people with schizophrenia consistently reveal enlarged ventricles. The brain has four ventricles, which are spaces in the brain filled with cerebrospinal fluid. Having larger fluid-filled spaces in and around the brain implies a loss of brain cells. Meta-analyses of neuroimaging studies have revealed that some people with schizophrenia, even very early in the course of the illness and across the course of the illness, have enlarged ventricles (Kempton, Stahl, Williams, & DeLisi, 2010; Olabi, Ellison-Wright, McIntosh et al., 2011; Wright et al., 2000). Further evidence concerning enlarged ventricles comes from a meta-analysis of 69 studies that included over 2000 people with schizophrenia who had never taken antipsychotic medication (Haijma et al., 2013). This is important because it helps to rule out the possibility that enlarged ventricles or other overall brain volume reductions may be due to the adverse effects of medications. Two MRI studies of MZ twin pairs, only one of whom had schizophrenia, assessed ventricle size (McNeil, Cantor-Graae, & Weinberger, 2000; Suddath et al., 1990). In both studies the ill twins had larger ventricles than the well twins, and in one of the studies most of the twins with schizophrenia could be identified by simple visual inspection of the scan. Because the twins were nearly genetically identical in these studies, these results suggest that the origin of enlarged ventricles may not be genetic.

Large ventricles in people with schizophrenia are correlated with impaired performance on neuropsychological tests, poor functioning prior to the onset of the disorder and poor response to medication treatment (Andreasen, Olsen, Dennert, & Smith, 1982; Weinberger, Cannon-Spoor, Potkin, & Wyatt, 1980). The extent to which the ventricles are enlarged, however, is modest, and many people with schizophrenia do not differ from people without schizophrenia in this respect. Furthermore, enlarged ventricles are not specific to schizophrenia, as they are also evident in the CT scans of people with other disorders, such as bipolar disorder with psychotic features (Rieder, Mann, Weinberger, van Kammen, & Post, 1983). People with these disorders can show ventricular enlargement almost as great as that seen in schizophrenia (Elkis, Friedman, Wise, & Meltzer, 1995). Other findings also suggest that perhaps the schizophrenia and psychotic mood disorders should not be totally separate diagnostic categories. The disorders share some symptoms (notably, delusions) and some possible aetiological factors (e.g., genetic factors, increased dopamine activity) and they respond similarly to medications. Researchers would be well served to focus some of their efforts on psychotic symptoms in other disorders as well as in schizophrenia.

### Factors involving the prefrontal cortex

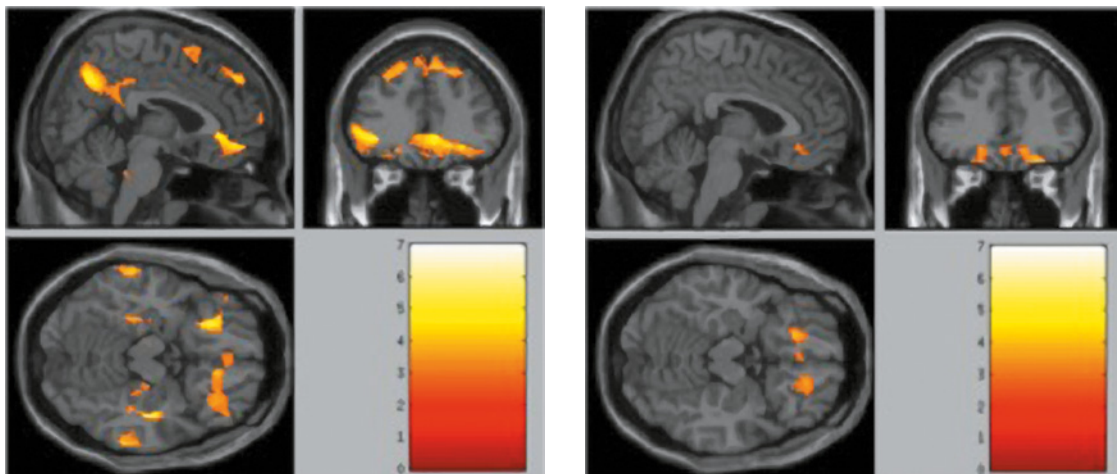
A variety of evidence suggests that the prefrontal cortex is of particular importance in schizophrenia.

- The prefrontal cortex is known to play a role in behaviours such as speech, decision making, emotion and goal-directed behaviour, which are disrupted in schizophrenia.
- MRI studies have shown reductions in grey matter and overall volume in the prefrontal cortex (Ohtani et al., 2014; Bora et al., 2011). These changes are associated with the transition to psychosis in

people with an at-risk mental state or APS (Sun et al., 2009). The duration of relapse of psychosis is negatively correlated with frontal lobe and total brain volume (Andreasen, Liu, Ziebell, Vora, & Ho, 2013).

- People with schizophrenia perform more poorly than people without schizophrenia on neuropsychological tests designed to tap functions supported by the prefrontal region, including working memory or the ability to hold bits of information in memory (Barch, Csernansky, Conturo, & Snyder, 2002; Barch, Carter, MacDonald, Braver, & Cohen, 2003; Heinrichs & Zakzanis, 1998), with some evidence that performance on some of these tests declines from before disorder onset to when people are in their late 30s (Meier, Caspi, Reichenberg et al., 2014).
- PET brain-imaging studies find that people with schizophrenia show lower glucose metabolism in the prefrontal cortex when performing neuropsychological tests tapping prefrontal function (Buchsbbaum, Kessler, King, Johnson, & Cappelletti, 1984). Because the tests place demands on the prefrontal cortex, glucose metabolism normally goes up as energy is used. People with schizophrenia, especially those with prominent negative symptoms, do poorly on the tests and also show less activation in the prefrontal region (Potkin, Alva, Fleming et al., 2002; Weinberger, Berman, & Illowsky, 1988). People with schizophrenia also show less blood flow to the brain as indicated with fMRI during performance of these same types of tests (Barch, Carter, Braver et al., 2001; MacDonald & Carter, 2003).
- Finally, failure to show frontal activation is related to the severity of negative symptoms (O'Donnell & Grace, 1998; Ohtani et al., 2014) and thus parallels the work on dopamine underactivity in the frontal cortex already discussed.

The pictures show brain activation from an fMRI study that involved maintaining pleasant emotional experience over a 12-second delay. The control group showed greater activation in areas of the frontal lobes compared to the schizophrenia group.

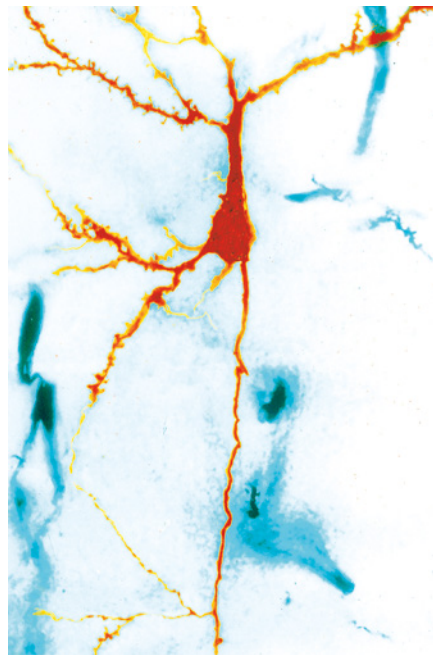


Despite the reduced volume of the grey matter in the prefrontal cortex (and also the temporal cortex), the number of neurons in this area does not appear to be reduced. More detailed studies indicate that what is lost may be what are called ‘dendritic spines’ (Glausier & Lewis, 2013; Goldman-Rakic & Selemon, 1997; McGlashan & Hoffman, 2000). Dendritic spines are small projections on the shafts of dendrites where nerve impulses are received from other neurons at the synapse (see figure 6.6). The loss of these dendritic spines means that communication among neurons (i.e., functioning of the synapses) is disrupted, resulting in what some have termed a ‘disconnection syndrome’ (Friston, Brown, Siemerku, & Stephan, 2016). One possible result of the failure of neural systems to communicate could be the speech and behavioural disorganisation seen in schizophrenia. Current research is linking these abnormalities in dendritic spines with the candidate genes and CNVs

identified in schizophrenia (Pathania et al., 2014; Penzes, Cahill, Jones, VanLeeuwen, & Woolfrey, 2011). It has also identified a possible mechanism for the excessive pruning of synapses that probably underlies this change in connectivity via the aberrant activation of complement. The complement cascade is an essential immune response to pathogens and cellular debris. Genetic variation in the C4 component of this cascade has been associated with the accelerated destruction of synapses in the brain possibly by prompting microglia to aggressively phagocytose these synapses (Sekar et al., 2016).

**FIGURE 6.6**

Micrograph of a neuron. The bumps on the dendrites are dendritic spines, which receive inputs from other neurons. Subjects with schizophrenia have fewer dendritic spines, impairing connectivity among neurons.



*Source:* Glantz & Lewis (2000).

### Problems in the temporal cortex and surrounding regions

Additional research has found that people with schizophrenia have structural and functional abnormalities in the temporal cortex, including areas such as the temporal gyrus, hippocampus, amygdala and anterior cingulate. For example, research also shows a reduction in cortical grey matter in temporal as well as frontal brain regions (Gur, Turetsky, Cowell et al., 2000) and reduced volume in the basal ganglia (e.g., the caudate nucleus), hippocampus and limbic structures (Keshavan, Rosenberg, Sweeney, & Pettegrew, 1998; Lim et al., 1998; Mathew et al., 2014; Nelson, Saykin, Flashman, & Riordin, 1998; Velakoulis et al., 1999). A twin study found reduced hippocampus volume among twins with schizophrenia, but not among the twins without schizophrenia (van Erp, Saleh, Huttunen et al., 2004). A meta-analysis of MRI studies conducted with people during their first episode of schizophrenia concluded that the volume of the hippocampus was significantly reduced compared to people without schizophrenia (Steen, Mull, McClure, Hamer, & Lieberman, 2006).

An additional interesting piece of evidence regarding the hippocampus comes from a meta-analysis of nine studies assessing the brain volume of over 400 first-degree relatives of people with schizophrenia and over 600 first-degree relatives of people without schizophrenia (Boos, Aleman, Cahn, Hulshoff, & Kahn, 2007). Relatives of people with schizophrenia had smaller hippocampal volumes than relatives of

people without schizophrenia. These findings suggest that reduced hippocampal volume in people with schizophrenia may reflect a combination of genetic and environmental factors.

What makes these findings about the hippocampus all the more intriguing is the fact that the hypothalamic–pituitary–adrenal (HPA) axis is closely connected to this area of the brain. Chronic stress is associated with reductions in hippocampal volume in other disorders, such as post-traumatic stress disorder (see a previous chapter). Although people with schizophrenia do not necessarily experience more stress than people without schizophrenia, they are more reactive to stress. Other evidence indicates that the HPA axis is disrupted in schizophrenia, particularly very early in the course of the disorder (Walker et al., 2008; Walker et al., 2013). Taken together, stress reactivity and a disrupted HPA axis likely contribute to the reductions in hippocampal volume observed in people with schizophrenia (Walker et al., 2008).

## Connectivity in the brain

Recent neuroimaging methods measure how different areas of the brain are connected to each other. Given the widespread brain dysfunction in schizophrenia, it is perhaps not surprising that the connections between areas are also problematic (Rapoport, Giedd, & Gogtay, 2012). Understanding the interconnectedness of the brain allows us to view changes in function as not bound by a single locus but reflecting a network response that is more or less adaptive (Fornito, Zalesky, & Breakspear, 2015). Also, as many of the hypothesised causes of the disorder originate at early stages in the development of the person, it is possible that schizophrenia occurs as the result of long-term neurodevelopmental changes in the ‘connectome’ of the brain (Crossley et al., 2014; Fornito, Zalesky, & Breakspear, 2015).

Broadly speaking, there are three types of connectivity. *Structural (or anatomical) connectivity* refers to how different structures of the brain are connected via white matter (axon fibres). Studies have found that people with schizophrenia have less connectivity in brain white matter than people without schizophrenia in the frontal and temporal cortices (Ellison-Wright & Bullmore, 2009).

*Functional connectivity* refers to the connectivity between brain regions based on correlations between their blood oxygen level dependent (BOLD) signal measured with fMRI (see chapter 1). Several studies have found reduced functional connectivity in schizophrenia, particularly in the frontal cortex (Pettersson-Yeo, Allen, Benetti, McGuire, & Mechelli, 2011).

*Effective connectivity* combines both types of connectivity in that it not only reveals correlations between BOLD activations in different brain regions but also the direction and timing of those activations by showing, for example, that activation in the occipital cortex comes first, followed by activation on the frontal cortex when someone is viewing pictures of objects (Friston, 1994). Like the other types of connectivity, research has found diminished effective connectivity in schizophrenia (Das et al., 2007; Deserno, Sterzer, Wustenberg, Heinz, & Schlagenhauf, 2012).

These connectivity methods have revealed a number of what are called brain networks. Networks are clusters of brain regions that are connected to one another in that activation in these regions is reliably correlated when people perform certain types of tasks or are at rest. For example, the frontoparietal network involves activation of the frontal and parietal cortices and this network is activated when people are doing cognitive tasks. The default-mode network involves areas of the prefrontal cortex and temporal cortex and is activated when people are daydreaming or thinking about the future and recalling memories.

Other brain connectivity research in schizophrenia has revealed that there is less connectivity between brain networks, including the frontoparietal and default-mode networks and this diminished connectivity is correlated with poor performance on cognitive tests (Unschuld et al., 2014; Fornito, Yoon, Zalesky, Bullmore, & Carter, 2011). Research has also found diminished connectivity among healthy relatives of people with schizophrenia, suggesting that diminished connectivity might be part of the genetic diathesis for schizophrenia (Collin, Kahn, de Reus, Cahn, & van den Heuvel, 2014; Unschuld et al., 2014).

## 6.4 Environmental factors influencing the developing brain

**LEARNING OUTCOME 6.4** Describe the role of environmental factors in the aetiology and relapse of schizophrenia.

Several different environmental factors have been studied as possible contributing factors to schizophrenia (Brown, 2011; van Os, Kenis, & Rutten, 2010). A possible cause of some of the observed brain abnormalities in schizophrenia is damage during gestation or birth. Many studies have shown high rates of delivery complications in people with schizophrenia (Brown, 2011; Walker, Kestler, Bollini, & Hochman, 2004); such complications could have resulted in a reduced supply of oxygen to the brain, resulting in loss of cortical grey matter (Cannon, van Erp, Rosso et al., 2002). These obstetrical complications do not raise the risk for schizophrenia in everyone who experiences them. Rather, the risk for schizophrenia is increased in those who experience complications and have a genetic diathesis (Cannon & Mednick, 1993).

Starvation has also been associated with an increased risk of schizophrenia. This particular association was recognised in two separate cohorts of people. The original cohort experienced the famine associated with the end of the Second World War in the Netherlands. Over the winter of 1944–45, during the period of chaos associated with the collapse of Hitler's Germany, a large section of the Dutch population was starved with a dietary intake of, on average, less than 1000 kcal (4200 kJ) daily. The effect upon fetuses during the first trimester of development was marked with a doubling of the risk of developing schizophrenia (Susser et al., 1996). This unfortunate association was replicated in a population cohort in China starved during the famine of 1959–61 (St Clair et al., 2005).

Additional research suggests that maternal infections during pregnancy are associated with greater risk of their children developing schizophrenia when they become adults (Brown & Derkits, 2010). For example, one study found that maternal exposure to the parasite *Toxoplasma gondii* was associated with a nearly 2.5 times greater risk of schizophrenia among the mothers' children when they became adults (Brown et al., 2005). This is a common parasite, carried by many people with no ill effects.

The most widely studied prenatal infection has been influenza. Studies have examined rates of schizophrenia among adults who had likely been exposed to the flu virus during their mothers' pregnancies (Mednick, Machon, Huttunen, & Bonett, 1988; Mednick, Huttunen, & Machon, 1994). During the 1957 Helsinki influenza epidemic, people who had been exposed to the flu virus during the second trimester of pregnancy had much higher rates of schizophrenia than those who had been exposed in either of the other trimesters and much higher rates than non-exposed control adults. This finding was replicated in only half of nearly 30 later studies, calling it into question. A more recent study found evidence that mothers' exposure to the flu during the *first* trimester of pregnancy, as directly measured by the presence of flu antibodies in the blood, was associated with a sevenfold increase in the risk for schizophrenia among their children (Brown et al., 2004). Although the increase in risk sounds large, the difference from the control group was not quite statistically different, suggesting it is a small effect.

If, as the findings we have just reviewed suggest, the development of the brains of people with schizophrenia goes awry very early, why does the disorder begin many years later, in adolescence or early adulthood? The prefrontal cortex is a brain structure that matures late, typically in adolescence or early adulthood. Thus, a problem in this area, even one that begins early in the course of development, may not show itself in the person's behaviour until the period of development when the prefrontal cortex begins to play a larger role in behaviour (Howes, McCutcheon, Owen, & Murray, 2017; Weinberger, 1987). Notably, dopamine activity also peaks in adolescence, which may further set the stage for the onset of schizophrenia symptoms (Walker et al., 2008). Adolescence is also typically a developmental period that is fraught with stress. Recall from our discussions in chapter 1 that stress activates the HPA axis, causing cortisol to be secreted. Research in the past 10 years has demonstrated that cortisol increases dopamine activity, particularly in the mesolimbic pathway, perhaps increasing the likelihood of developing schizophrenia symptoms (Walker et al., 2008).

Another proposed explanation is that the development of symptoms in adolescence could reflect a loss of synapses due to excessive pruning, the elimination of synaptic connections. Pruning is a normal part of brain development that occurs at different rates in different areas of the brain. It is mostly complete in sensory areas by about two years of age but continues in the prefrontal cortex until mid-adolescence. If too extensive, pruning may result in the loss of necessary communication among neurons (McGlashan & Hoffman, 2000). This reduction in synaptic connections can be seen in the prefrontal cortex in schizophrenia (Glantz & Lewis, 2000) and recent evidence associating a genetically mediated increase in complement activity which marks synapses requiring elimination by microglia in the brain has provided a physiologically based mechanism for this to occur (Sekar et al., 2016) — however, this has not been seen during the critical period of the development of psychosis as yet.

An additional environmental factor that has been studied as a risk factor for schizophrenia among adolescents is cannabis (marijuana) use. Among people already diagnosed with schizophrenia, cannabis use is associated with a higher level of positive symptoms, depression and poorer functioning, particularly in people early in the course of their illness (Mullin et al., 2012). But does cannabis use contribute to the onset of schizophrenia? A longitudinal study examining the prospective relationship between cannabis use in adolescence and the onset of schizophrenia in adolescence or adulthood indicated that the risk of developing schizophrenia symptoms was greater among those who used cannabis compared to those who did not (Arseneault et al., 2002). This was backed up by a meta-analysis of 83 studies that found that cannabis users had an earlier age of onset for psychosis (Large, Sharma, Compton, Slade, & Nielssen, 2011). Furthermore, more frequent use and higher potency cannabis are associated with greater risk (Di Forti et al., 2013). However, correlation does not mean causation. Other studies suggest that the linkage between cannabis use and risk of developing schizophrenia is observed only among those who are genetically vulnerable to schizophrenia. A gene-environment interaction has been observed with two genes of interest in schizophrenia. An increased risk of psychosis has been associated with a particular polymorphism in the *COMT* gene (Caspi et al., 2005) and the *AKT1* gene (Di Forti et al., 2012). Both vulnerabilities required the abuse of cannabis to activate the risk associated with them.

## Psychological factors

People with schizophrenia do not appear to experience more stress in daily life than people without schizophrenia (Phillips, Francey, Edwards, & McMurray, 2007; Walker et al., 2008). However, people with the disorder appear to be very reactive to the stressors we all encounter in daily living. In one study, people with psychotic disorders (92 percent with schizophrenia), their first-degree relatives and people without any psychiatric disorder participated in a six-day ecological momentary assessment study in which they recorded stress and mood several times each day. Daily life stress predicted greater decreases in positive moods in both people with schizophrenia and their relatives compared with controls. Stress also predicted greater increases in negative moods in the people with schizophrenia compared with both relatives and controls (Myin-Germeys, van Os, Schwartz et al., 2001). Thus, people with schizophrenia were particularly vulnerable to daily stress. Research also shows that, as with many of the disorders we have discussed in this text, increases in life stress increase the likelihood of a relapse (Ventura, Neuchterlein, Lukoff, & Hardesty, 1989; Walker et al., 2008).

Additional research on psychological factors in the development and relapse of schizophrenia has focused on socioeconomic status and the family.

## Socioeconomic status

For many years we have known that schizophrenia can be found at all levels of socioeconomic status (SES) in many countries, but that the highest rates of schizophrenia are found in people with the lowest SES (Hollingshead & Redlich, 1958; Kohn, 1968).

The correlation between SES and schizophrenia is consistent but difficult to interpret in causal terms. Is it that the stress associated with poverty, such as low education, limited opportunities and stigma from others of high status contributes to the development of schizophrenia — the **sociogenic hypothesis**?

Or is it the case that during the course of their developing illness, people with schizophrenia drift into poor neighbourhoods because their illness impairs their earning power and they cannot afford to live elsewhere — the **social selection hypothesis**?

A study in Israel evaluated the two hypotheses by investigating both SES and ethnic background (Dohrenwend et al., 1992). The rates of schizophrenia were examined among Israeli Jews of European ethnic background and among more recent immigrants to Israel from North Africa and the Middle East. The latter group experienced considerable racial prejudice and discrimination in Israel. The sociogenic hypothesis would predict that because they experienced high levels of stress regardless of socioeconomic status, the members of the disadvantaged ethnic group should have consistently higher rates of schizophrenia regardless of status. However, this pattern did not emerge, supporting the social selection hypothesis. Thus, research results are more supportive of the social selection hypothesis than of the sociogenic hypothesis.

### Family-related factors

Early theorists regarded family relationships, especially those between a mother and her son, as crucial in the development of schizophrenia. At one time, the view was so prevalent that the terms *schizophrenogenic mother* was coined for the supposedly cold and dominant, conflict-inducing parent who was said to produce schizophrenia in her offspring (Fromm-Reichmann, 1948). Controlled studies evaluating the schizophrenogenic mother theory have not supported it. The damage done to families by this theory, however, was significant. For generations, parents blamed themselves for their child's illness and until the 1970s, psychiatrists often joined in this blame game.

#### How do families influence schizophrenia?

Other studies continued to explore the possibility that the family plays some role in the aetiology of schizophrenia. For the most part, the findings are only suggestive, not conclusive. For example, a few studies of families of people with schizophrenia have found that they communicate more vaguely with one another and have higher levels of conflict than families of people without schizophrenia. It is plausible, though, that the conflict and unclear communication are a response to having a young family member with schizophrenia.

#### Families and relapse

A series of studies initiated in London found that the family can have an impact on the recovery of people with schizophrenia after they leave the hospital. In one study, investigators conducted a nine-month follow-up study of a sample of people with schizophrenia who returned to live with their families after being discharged from the hospital (Brown, Bone, Dalison, & Wing, 1966). Interviews were conducted with parents or spouses before discharge and rated for the number of critical comments made about the family member with schizophrenia and for expressions of hostility towards and emotional overinvolvement with the ill relative. The following is an example of a critical comment made by a father remarking on his daughter's behaviour, in which he is expressing the idea that his daughter is deliberately symptomatic to avoid housework: 'My view is that Maria acts this way so my wife doesn't give her any responsibilities around the house' (quote in Weisman, Neuchterlein, Goldstein, & Snyder, 1998). Combining these three characteristics — critical comments, hostility and emotional overinvolvement — led to the creation of the construct called **expressed emotion (EE)**. Families in the original study were divided into two groups: those revealing a great deal of expressed emotion (high-EE families) and those revealing little (low-EE families). At the end of the follow-up period, only 10 percent of the people returning to low-EE homes had relapsed, but 58 percent of the people returning to high-EE homes had gone back to the hospital.

This research, which has since been replicated (see Butzlaff & Hooley, 1998, for a meta-analysis), indicates that the home environment of people with schizophrenia can influence how soon they relapse. Researchers have also found that negative symptoms of schizophrenia are most likely to elicit critical comments, as in the example presented in the previous paragraph and that the relatives who make the most critical comments are the most likely to view people with schizophrenia as being able to control their symptoms (Lopez, Nelson, Snyder, & Mintz, 1999; Weisman et al., 1998).

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Expressed emotion, which includes hostility, critical comments and emotional overinvolvement, has been linked with relapse in schizophrenia.



There are also important cultural differences in EE, however; the relationship between emotional overinvolvement and relapse in other European and Asian countries is not as robust as it is in North American countries (Singh, Harley, & Suhail, 2013). The different findings may be attributed either to the fact that emotional overinvolvement is measured differently across countries and cultures or that emotional overinvolvement is more harmful in some cultures and countries than in others. This issue needs to be sorted out in future research.

What is not yet clear is exactly how to interpret the effects of EE. Is EE causal or does it reflect a reaction to the ill relative's behaviour? For example, if the condition of a person with schizophrenia begins to deteriorate, family concern and involvement may increase. Indeed, disorganised or dangerous behaviour might seem to warrant familial efforts that could increase the level of EE. Research indicates that both interpretations of the operation of EE may be correct. In one study, people with schizophrenia and their high- or low-EE families were observed as they engaged in a discussion of a family problem. Two key findings emerged (Rosenfarb, Goldstein, Mintz, & Neuchterlein, 1994).

1. The expression of unusual thoughts by the people with schizophrenia ('If that kid bites you, you'll get rabies') elicited a greater number of critical comments by family members who had previously been characterised as high in EE than by those characterised as low in EE.
2. In high-EE families, critical comments by family members led to increased expression of unusual thoughts by the people with schizophrenia.

Thus, this study found a bidirectional relationship in high-EE families: critical comments by family members elicited more unusual thoughts by relatives with schizophrenia and unusual thoughts expressed by the relatives with schizophrenia led to increased critical comments.

How does stress, such as a high level of EE, increase the symptoms of schizophrenia and precipitate relapses? One answer to this question involves the effects of stress on the HPA axis and its link to

dopamine (Walker et al., 2008). Stress activates the HPA axis, causing cortisol to be secreted, which can then increase dopamine activity (Walker et al., 2008). Furthermore, heightened dopamine activity itself can increase HPA activation, which may make a person overly sensitive to stress. Thus, there is a bidirectional relationship between HPA activation and dopamine activity.

## Developmental factors

What are people who develop schizophrenia like before their symptoms begin? This question has been addressed using retrospective and prospective studies. The retrospective studies are sometimes referred to as 'follow-back' studies because the starting point is a group of adults with schizophrenia that researchers then follow back to childhood to unearth records and tests from their early years.

### Retrospective studies

In the 1960s, researchers found that children who later developed schizophrenia had lower IQs and were more often delinquent and withdrawn than members of various control groups, usually comprising siblings and neighbourhood peers (Albee, Lane, & Reuter, 1964; Berry, 1967; Lane & Albee, 1965). Other studies found that boys who later developed schizophrenia were rated by teachers as disagreeable, whereas girls who later developed schizophrenia were rated as passive (Watt, 1974; Watt, Stolorow, Lubensky, & McClelland, 1970).

More recently, researchers have examined emotional and cognitive deficits that were present before the onset of schizophrenia. In one study, Elaine Walker and colleagues analysed the home movies of children who later developed schizophrenia. The movies were made as part of normal family life and were made before the onset of schizophrenia (Walker, Davis, & Savoie, 1994; Walker, Grimes, Davis, & Adina, 1993). Compared with their siblings who did not later develop schizophrenia, the children who later developed schizophrenia as young adults showed poorer motor skills and more expressions of negative emotions. Other studies have examined past childhood assessments of the cognition and intellectual functioning of adults who had schizophrenia. These studies found that adults with schizophrenia scored lower on IQ and other cognitive tests as children compared to adults without schizophrenia (Davis, Malmberg, Brandt, Allebeck, & Lewis, 1997; Woodberry, Giuliano, & Seidman, 2008).

As intriguing as these findings are, these studies were not necessarily designed with the intention of predicting the development of schizophrenia from childhood behaviour. Rather, they began with an adult sample of people with schizophrenia and then looked back at records and data collected from their childhoods to see if there were characteristics that distinguished them as young children.

### Prospective studies

A more recent prospective study identified childhood characteristics that were associated with the development of schizophrenia in early adulthood. In this ongoing study, a cohort of 1037 people have been intensively studied since their birth in Dunedin, New Zealand. Reichenberg and colleagues examined the trajectory of change in cognition between the ages of 7 and 32 using serially administered IQ tests. The researchers (Reichenberg et al., 2010) found that lower scores on the IQ test in childhood predicted the onset of schizophrenia in young adulthood, even after they controlled for low socioeconomic status (which is associated with lower IQ scores; see chapter 2). The group with schizophrenia also suffered a significant decline from 13 to 32 years of age across a range of cognitive tasks including full-scale IQ, speed of processing, executive skills, learning and motor function. This change was not accounted for by other factors such as substance use (Meier et al., 2014) and was not seen in other people with depression or a group with mild cognitive impairment. These findings are broadly consistent with the idea that something goes awry in development that is associated with the onset of schizophrenia in late adolescence or early adulthood. Nevertheless, more specific information is required if developmental histories are to provide clear evidence regarding the aetiology of schizophrenia.

One type of study used in schizophrenia research is the **familial high-risk study**, which begins with one or two biological parents with schizophrenia and follows their offspring longitudinally in order to

identify how many of these children may develop schizophrenia and what types of childhood neurobiological and behavioural factors may predict the disorder's onset. One of the difficulties with familial high-risk studies has to do with the large sample sizes that are required. As shown in table 6.2, around 10 percent of children with a biological parent who has schizophrenia go on to develop schizophrenia. If a study begins with 200 high-risk children, only about 20 of them may go on to develop schizophrenia. In addition, it is not particularly easy to locate a large sample of women or men with schizophrenia who have had their own children.

Because of these difficulties, the **clinical high-risk study** has been used in more recent research. A clinical high-risk study is a design that identifies people with early, attenuated signs of schizophrenia, most often milder forms of hallucinations, delusions or disorganisation that nonetheless cause impairment (see focus on discovery 6.3). One such study followed people between the ages of 14 and 30 in Australia who were referred to a mental health clinic in the mid-1990s (Yung, McGorry, McFarlane, & Patton, 1995). None of the participants had schizophrenia when they entered the study, but many later exhibited varying degrees of schizophrenia symptoms and some, but not all, had a biological relative with a psychotic disorder. These participants were deemed to have an *at-risk mental state* (ARMS) and to be at *ultra-high risk* of developing schizophrenia or psychotic disorders. Since the study began, 114 of the 416 participants have developed some type of psychotic disorder mostly in the first two years after referral. However, the risk of transition to psychosis continued to extend for at least 10 years after referral (Nelson et al., 2013). Importantly, even if the individual didn't develop a psychotic disorder they were at very high risk of continuing to experience attenuated psychotic symptoms (28 percent) or of being diagnosed with another mental disorder (68.1 percent) (Lin et al., 2015). This suggests that this group of individuals is at very high risk of illness and functional impairment, supporting the validity of this approach to identifying young people with significant mental health issues. An MRI study of 75 of the 104 participants found that those people who later developed a psychotic disorder had smaller grey matter volumes than those who had not developed a psychotic disorder (Pantelis, Velakoulis, McGorry et al., 2003). Recall that reduced grey matter volume has been found in people with schizophrenia; the Pantelis et al. (2003) study suggests that this characteristic may predate the onset of schizophrenia and other psychotic disorders.

This approach to the early identification of people at risk of developing a psychotic disorder such as schizophrenia has been replicated internationally. A meta-analysis of the 27 studies that had adopted variants of the ARMS criteria (Fusar-Poli et al., 2012) found a transition risk of 29.1 percent at two years, rising to 35.8 percent at more than three years. The criteria appeared to be more effective in identifying people developing a schizophrenia spectrum disorder rather than a psychotic mood disorder (Fusar-Poli et al., 2012), prompting attempts to define similar criteria that are sensitive to psychotic mood disorders such as bipolar disorder (Bechdolf et al., 2010).

## 6.5 Treatment of schizophrenia

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**LEARNING OUTCOME 6.5** Discuss the appropriate use of medication and psychological treatments for schizophrenia.

Treatments for schizophrenia most often include a combination of short-term hospital stays (during the acute phases of the illness), medication and psychosocial treatment. A problem with treatment is that some people with schizophrenia lack insight into their impaired condition and may refuse treatment — both medication and psychological treatment (Amador et al., 1994). Results from one study suggest that gender (female) and age (older) are predictors of better insight among people in their first episode of the illness (McEvoy et al., 2006) and this may help account for why women with schizophrenia tend to respond better to treatment than men (Salem & Kring, 1998). Those who lack insight, and thus don't believe they have an illness, don't see the need for professional help, particularly when it includes hospitalisation or drugs. Family members therefore face a major challenge in getting their relatives into treatment.

## Medications

In the 1950s, several medications collectively referred to as *antipsychotic drugs* (also referred to as *neuroleptics* or *major tranquillisers*) were found to help with the positive symptoms and disorganisation of schizophrenia. For the first time, many people with schizophrenia did not need to stay in a hospital for long periods of time and could instead go home with a prescription. Focus on discovery 6.4 presents a brief history of the development of these drugs. The zeal to discharge people with schizophrenia from hospitals did not match the needs of all people with the disorder. Some people did and still do need treatment in a hospital, even if for a short time. Unfortunately, it is difficult to receive such treatment today due to the cost and limited availability of hospital beds for people with schizophrenia and other severe mental disorders. Nevertheless, medications have made it possible for most people with schizophrenia to lead lives outside of a hospital. However, by themselves they are not sufficient and, as we will see, medications have their own drawbacks. In Australia, 81.6 percent of people with a psychotic illness are taking an antipsychotic medication (Morgan et al., 2011), the vast majority taking a second-generation antipsychotic medication.

### FOCUS ON DISCOVERY 6.4

#### Stumbling towards a cure: the development of antipsychotic medications

The earliest effective antipsychotic was probably reserpine, an extract from *Rauwolfia serpentina*, an ancient Indian and Chinese herbal medicine. However, it was not until the use of chlorpromazine by Delay and Deniker in France in 1952 that an effective antipsychotic was found.

Chlorpromazine was synthesised in 1951 originally to potentiate anaesthetics in surgery. A French surgeon, Henri Laborit, noticed that it and other histamine-like compounds made his patients somewhat sleepy and less fearful about the impending operation, and he suggested that there may be a use for the new medication in psychiatry. This drug, from a class of drugs called phenothiazines, proved very effective in calming people with schizophrenia. Phenothiazines derive their antipsychotic properties from blocking dopamine receptors in the brain, thus reducing the influence of dopamine on thought, emotion and behaviour. They were rapidly complemented by the synthesis of other phenothiazines (e.g., trifluoperazine) and butyrophenones (e.g., haloperidol), all of which had this common property of dopamine blockade.

However, with the widespread use of the antipsychotic medications for the treatment of schizophrenia it became obvious that this first generation of antipsychotic medications caused a wide variety of adverse effects matching their broad range of receptor blockade, such as involuntary movements of the face, arms and legs (dopaminergic), sedation (histaminic), weight gain (range of receptor and hormonal effects) and dry mouth or blurry vision (anticholinergic). It also became clear that they did not treat all of the symptoms of schizophrenia and that, in particular, the negative and cognitive symptoms were at best only partially treated. This has led to further drug development and the introduction of the second-generation antipsychotics that were thought to have broader neurotransmitter effects, such as effects upon the serotonin 5-HT<sub>2A</sub> receptor.

#### QUESTIONS

1. Which groups of symptoms do first-generation antipsychotics best treat?
2. Why do antipsychotics cause movement disorders?

Antipsychotic medication can be administered as tablets or capsules, or in an injectable form. Injectable medications can be given for immediate short-term effect or as a long-acting injectable (LAI) form that may last for up to three months. The LAIs have been developed particularly in light of the poor treatment adherence of many people with schizophrenia. About 30 percent of people with schizophrenia do not respond favourably to the first-generation antipsychotics; about half the people who take any antipsychotic drug quit after one year, and up to three-quarters quit before two years (Lieberman et al., 2005). Although

the adverse effects of antipsychotic medications are one of the key drivers of this, poor insight into illness, beliefs about medication and substance abuse are also important factors (Higashi et al., 2013).

### First-generation antipsychotic drugs and their adverse effects

The first-generation antipsychotic drugs are those broad classes of medications that were the first to be discovered. See table 6.4 for a summary of major drugs used to treat schizophrenia. These drugs can reduce the positive and disorganisation symptoms of schizophrenia but have little or no effect on the negative and cognitive symptoms, perhaps because their primary mechanism of action involves blocking dopamine D2 receptors. Despite the enthusiasm with which these drugs were prescribed, they are not a cure.

**TABLE 6.4** Summary of major drugs used in treating schizophrenia

Drug category	Generic name	Trade name**
<b>First-generation drugs</b>	chlorpromazine	Largactil
	fluphenazine decanoate*	Modecate
	flupenthixol decanoate*	Fluanxol
	haloperidol*	Seranace, Haldol
	trifluoperazine	Stelazine
	zuclopenthixol decanoate*	Clopixol
<b>Second-generation drugs</b>	amisulpride	Solian
	aripiprazole*	Abilify
	asenapine	Saphris
	clozapine	Clozaril, Clopine
	lurasidone	Latuda
	olanzapine*	Zyprexa
	paliperidone*	Invega
	quetiapine	Seroquel
	risperidone*	Risperdal
	ziprasidone	Zeldox

\*Also available as a LAI or as a LAI only

\*\*A variety of different trade names may exist

People who respond positively to the antipsychotics are typically kept on so-called maintenance doses of the drug to continue the therapeutic effect. Results from a meta-analysis of over 60 randomised controlled clinical trials comparing either first- or second-generation drugs to placebo found that maintenance dosages of both were equally effective at reducing relapse compared to placebo (Leucht et al., 2009). Some people who are maintained on medication may still have difficulty with day-to-day

functioning, however. For example, they may be unable to live independently or to hold down the kind of job for which they would otherwise be qualified, and their social relationships may be few. In short, some symptoms may be controlled, but lives are still not fulfilling for many people with schizophrenia.

The commonly reported adverse effects of all antipsychotics include sedation, dizziness, blurred vision, restlessness and sexual dysfunction. In addition, some particularly disturbing adverse effects, termed *extrapyramidal adverse effects*, resemble the symptoms of Parkinson's disease. People taking antipsychotics may develop tremors of the hands and fingers, a shuffling gait, a blank mask-like face and drooling. Other adverse effects can include dystonia, a state of muscular rigidity or spasm, and dyskinesia, an abnormal motion of voluntary and involuntary muscles, producing chewing movements of the tongue and jaw as well as other movements of the lips, fingers and legs. Another adverse effect is akathisia, an inability to remain still; people who have this adverse effect pace constantly and fidget.

In a neurological disturbance called *tardive dyskinesia*, the mouth muscles involuntarily make sucking, lip-smacking and chin-wagging motions. In more severe cases, the whole body can be subject to involuntary motor movements. This syndrome is observed mainly in older people with schizophrenia who had been treated with first-generation medications before drugs were developed to prevent tardive dyskinesia. It affects about 10 to 20 percent of these older people treated with first-generation antipsychotics for a long period of time and is not responsive to any known treatment (Sweet et al., 1995). Finally, an adverse effect called *neuroleptic malignant syndrome* occurs in about 1 percent of cases. In this condition, which can sometimes be fatal, severe muscular rigidity develops, accompanied by fever. The heart races, blood pressure is unstable and the person may lapse into a coma.

Because of these serious adverse effects, current Australian clinical practice guidelines advise that the lowest effective dose of medication be given and that close attention be given to the balance of beneficial effect as against adverse effect for the individual (Galletly et al., 2016).

## **Second-generation antipsychotic drugs and their adverse effects**

Considerable treatment resistance to existing antipsychotic medications led to the reuse of an antipsychotic called clozapine that had been removed from use because of its propensity to cause agranulocytosis — a potentially fatal adverse effect where infection-fighting white blood cells or neutrophils are reduced in number and the person becomes vulnerable to infection. Clozapine was known to be effective in people with schizophrenia who had not responded to other antipsychotic treatments, and this was demonstrated in 1988 in a landmark randomised, closely controlled trial against chlorpromazine (Kane, Honigfeld, Singer, Meltzer, & Clozaril Collaborative Study, 1988). Clozapine was also known to affect a very wide range of neurotransmitter systems, and this stimulated the introduction of a new generation of antipsychotic medications.

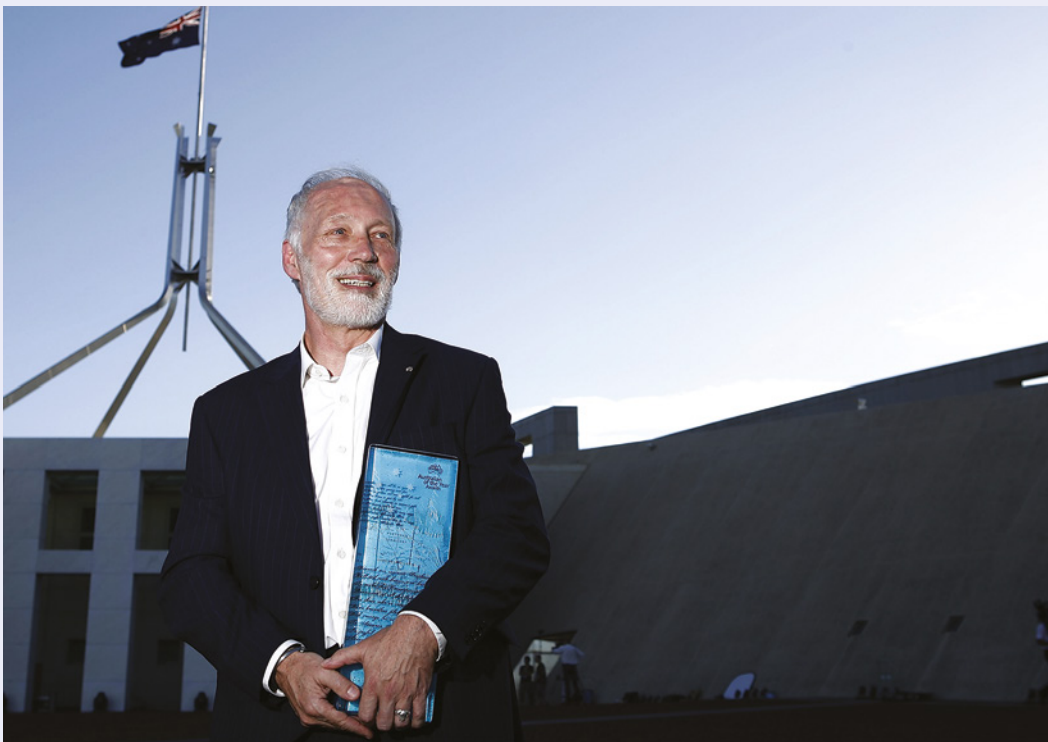
These drugs, including clozapine, are referred to as the **second-generation antipsychotic drugs** because their mechanism of action is not like that of the typical or first-generation antipsychotic medications. However, they do not form a single pharmacological class and really could be called 'new' as against 'old' antipsychotic medications. These newer antipsychotic medications have brought a range of advantages, but these have needed to be traded off against their own adverse effects.

Direct comparisons between the different antipsychotic medications are relatively limited; however, a series of meta-analyses suggest that there are differences between the medications (Kishimoto et al., 2013; Leucht, Arbter, Engel, Kissling, & Davis, 2008; Leucht et al., 2013; Leucht et al., 2009). Clozapine remains the most effective of the antipsychotic medications (Leucht et al., 2013; McEvoy et al., 2006), but after that there is a complex trade-off between the adverse effects of the medications and the individual profile of the person with schizophrenia. These adverse effects are many and include weight gain (especially prominent for clozapine and olanzapine), extrapyramidal adverse effects (in first-generation antipsychotics); increased secretion of prolactin (first-generation antipsychotics, amisulpride, risperidone and paliperidone); sedation (clozapine and chlorpromazine) and a prolonged QTc interval — a heart rhythm change (amisulpride and ziprasidone). Despite this formidable list of adverse effects, there is an advantage to the use of the second-generation antipsychotics over first-generation antipsychotics. People do prefer taking them (Lieberman et al., 2005) and there is a lower relapse rate associated with their use (Kishimoto et al., 2013).

Given the burden of adverse effects with antipsychotic medications, a number of alternate dosing strategies — including ‘drug holidays’ and early cessation of use — have been studied. Although a higher relapse rate is associated with drug holidays, the long-term outcome of people who have had a trial of medication cessation early in the course of their illness may be better, even though they require the reinstatement of medication. A long-term follow-up of a group of people with schizophrenia who had been randomised between treatment reduction or discontinuation and continued treatment found a higher rate of recovery in those allocated to the treatment discontinuation arm of the trial, even though they had a higher rate of relapse initially (Wunderink, Nieboer, Wiersma, Sytema, & Nienhuis, 2013).

#### RESEARCH EXAMPLE

##### Professor Patrick McGorry



Professor Patrick McGorry revolutionised the treatment of young people with schizophrenia by asking a simple question — why should we be intervening so late in the course of schizophrenia? During his early experience he noted how long it took for many people with psychosis to come to care and, once they had, how inappropriately they were treated in depressing psychiatric institutions without hope or age-appropriate care. His original unit EPPIC — the Early Psychosis Prevention and Intervention Centre — internationally pioneered the aggressive community identification and treatment of young people with psychosis. His team went on to develop the original criteria to identify people with an at-risk mental state for developing psychosis and have broadened their research and treatment agenda to include many aspects of youth mental health at Orygen, the National Centre of Excellence in Youth Mental Health ([www.orygen.org.au](http://www.orygen.org.au)).

#### QUESTION

What are the benefits of early diagnosis and treatment of young people with schizophrenia?

## Evaluation of drug treatments

Antipsychotic drugs are an indispensable part of treatment for schizophrenia and will undoubtedly continue to be so. Furthermore, the mixed success of second-generation antipsychotic drugs has stimulated a continued effort to find new and more effective drug therapies for schizophrenia. New drugs are currently being evaluated, but no significant breakthrough medications have yet been developed, nor medication that can treat psychosis through non-dopaminergic pathways. Thus, the ‘third generation’ is a way off.

## Psychological treatments

The limits of antipsychotic medications have spurred efforts to develop psychosocial treatments that can be used in addition to the medications. Indeed, the current treatment recommendations for schizophrenia emphasise the importance of combining medications and psychosocial interventions (Galletly et al., 2016; Kreyenbuhl, Buchanan, Dickerson, & Dixon, 2010; National Collaborating Centre for Mental Health, 2014). A number of psychosocial interventions, including skills training, cognitive-behavioural therapy, cognitive remediation therapy and family-based treatments, have a solid evidence base to support their use as an adjunctive treatment to medications (Dixon et al., 2010; Wykes, Huddy, Cellard, McGurk, & Czobor, 2011). A review of 37 prospective studies of people after their first episode of schizophrenia found that the combination of medication and psychosocial treatment predicted the best outcome (Menezes, Arenovich, & Zipursky, 2006).

An example of the positive effects that come from combination treatments is found in a large (over 1200 people) randomised controlled trial conducted in China that compared medication alone with medication plus a comprehensive psychosocial intervention that included family therapy, cognitive-behavioural therapy, psychoeducation and skills training. People in both groups had a similar reduction in schizophrenia symptoms. However, people who received the combined treatment had lower rates of relapse and treatment discontinuation, as well as greater improvements in functioning (Guo et al., 2010). See focus on discovery 6.5 for another example of a successful combination treatment approach.

Other treatments, such as cognitive remediation approaches, have a growing evidence base and are the focus of much current research. We turn to these psychosocial treatments next.

### Social skills training

**Social skills training** is designed to teach people with schizophrenia how to successfully manage a wide variety of interpersonal situations — discussing their medications with their psychiatrist, ordering meals in a restaurant, filling out job applications, interviewing for jobs, saying no to drug dealers on the street and reading bus schedules. Most of us take these skills for granted and give little thought to them in our daily lives, but people with schizophrenia cannot consider them a given — they need to work hard to acquire or reacquire such skills (Heinssen, Liberman, & Kopelowicz, 2000; Liberman, Eckman, Kopelowicz, & Stolar, 2000). Social skills training typically involves role-playing and other group exercises to practise skills, both in a therapy group and in actual social situations.

Research has shown that social skills training can help people with schizophrenia achieve fewer relapses, better social functioning and a higher quality of life (Kopelowicz, Liberman, & Zarate, 2002). Some of the studies are noteworthy in demonstrating benefits over a two-year period following treatment (Liberman, Wallace, Blackwell et al., 1998; Marder et al., 1999), though not all results are positive (Pilling et al., 2002). Social skills training is usually a component of treatments for schizophrenia that go beyond the use of medications alone, including family therapies for lowering expressed emotion, which we discuss next. For example, social skills training that included family therapy was found to be more effective than treatment as usual (medication plus a 20-minute monthly meeting with a psychiatrist) in a randomised controlled trial conducted in Mexico (Valencia, Racon, Juarez, & Murow, 2007). There is some evidence that social skills training may also be effective in reducing negative symptoms (Elis, Caponigro, & Kring, 2013).

## Living with schizophrenia

A heartening example of one woman's struggles with and triumphs over schizophrenia is found in the 2007 book *The Center Cannot Hold: My Journey Through Madness*. This book was written by Elyn Saks, an endowed professor of law at the University of Southern California who also happens to have schizophrenia (Saks, 2007). In the book, she describes her lifelong experience with this illness. Prior to the publication of the book, only a few of Professor Saks's close friends even knew that she had schizophrenia. Why did she keep it a secret? Certainly, stigma is part of the reason. As we have discussed throughout this book, stigma towards people with psychological disorders is very much alive in the twenty-first century and can have seriously negative consequences for people with disorders such as schizophrenia.

What makes Professor Saks's life story particularly encouraging is that she has achieved exceptional professional and personal success in her life despite having such a serious psychological disorder. She grew up in a loving and supportive family, earned a bachelor's degree from Vanderbilt University, graduating as her class valedictorian, earned a prestigious Marshall fellowship to study philosophy at Oxford in the United Kingdom, graduated from Yale Law School as editor of the prestigious *Yale Law Review* and is a tenured professor of law at a major university. How did she do it?

She believes that a combination of treatments (including psychoanalysis and medications), social support from family and friends, hard work and acknowledgement of the seriousness of her illness have all helped her cope with schizophrenia and its sometimes unpredictable and frightening symptoms. Although psychoanalysis does not have a good deal of empirical support for its efficacy with schizophrenia, it was and remains a central part of Professor Saks's treatment regimen. Thus, even though some treatments may not be effective for a group of people, they can nonetheless be beneficial for individuals. One characteristic that appears to have been helpful for Professor Saks, from her early days in psychoanalysis as a Marshall scholar at Oxford University until the present, has been her ability to 'be psychotic' when she is with her psychoanalyst. So much of her energy was spent trying to hide her symptoms and keep them from interfering with her life; psychoanalysis became a safe place for her to bring these symptoms more fully out into the open. The different analysts she has had over the years were also among the chief proponents of adding antipsychotic medication to her treatment, something that Professor Saks resisted for many years. Having the unwavering support of close friends and her husband has also been a tremendous help, particularly during her more symptomatic periods. Her loved ones would not turn and run the other way when she was psychotic. Instead, they would support her and help her get additional treatment if it was needed.

Professor Saks still experiences symptoms, sometimes every day. Her symptoms include paranoid delusions, which she describes as very frightening (e.g., believing that her thoughts have killed people). She also experiences disorganisation symptoms, which she eloquently describes in the book:

Consciousness gradually loses its coherence. One's center gives way. The center cannot hold. The 'me' becomes a haze and the solid center from which one experiences reality breaks up like a bad radio signal. There is no longer a sturdy vantage point from which to look out, take things in, assess what's happening. No core holds things together, providing the lens through which to see the world, to make judgments and comprehend risk. (Saks, 2007, p. 13)

Elyn Saks, a law professor at USC, has schizophrenia.



Even though she still experiences symptoms, she has come to terms with the fact that schizophrenia is a part of her life. Would she prefer not to have the illness? Sure. But she also recognises that she has a wonderful life filled with friends, loved ones and meaningful work. She is not defined by her illness and she importantly notes that 'the humanity we all share is more important than the mental illness we may not' (Saks, 2007, p. 336). Her life is an inspiration to all, not just those with psychological disorders. Her story reminds us that life is difficult, more so for some than others, but that it can be lived, and lived to the fullest.

### QUESTIONS

1. How has stigma affected Professor Saks' career? How has it manifested itself?
2. The majority of people with schizophrenia do not have the extent of recovery that Professor Saks has. What positive factors building her resilience can you see in her story?

## Family therapies

Many people with schizophrenia live with their families. Earlier we discussed research showing that high levels of expressed emotion (EE) within the family, including being hostile, critical and emotionally overinvolved, have been linked to relapse and rehospitalisation. Based on this finding, a number of family therapies have been developed. These therapies may differ in length, setting and specific techniques, but they have several features in common.

- *Education about schizophrenia.* Specifically, education about the genetic or neurobiological factors that predispose some people to the illness, the cognitive problems associated with schizophrenia, the symptoms of schizophrenia and the signs of impending relapse. High-EE families are typically not well informed about schizophrenia, and giving them some basic information helps them be less critical of the relative with schizophrenia. Knowing, for example, that neurobiology has a lot to do with having schizophrenia and that the illness involves problems in thinking clearly and rationally might help family members be more accepting and understanding of their relative's inappropriate or ineffectual actions. Therapists encourage family members to temper their expectations of their relative with schizophrenia, and they make clear to family and the person with schizophrenia alike that proper medication and therapy can reduce stress on the person and prevent deterioration.
- *Information about antipsychotic medication.* Therapists impress on both the family and the ill relative the importance of taking antipsychotic medication, becoming better informed about the intended effects and the adverse effects of the medication, taking responsibility for monitoring response to medication and seeking medical consultation rather than just discontinuing the medication if adverse effects occur.
- *Blame avoidance and reduction.* Therapists encourage family members to blame neither themselves nor their relative for the illness and for the difficulties all are having in coping with it.
- *Communication and problem-solving skills within the family.* Therapists focus on teaching the family ways to express both positive and negative feelings in a constructive, empathic, non-demanding manner rather than in a finger-pointing, critical or overprotective manner. They also focus on making personal conflicts less stressful by teaching family members ways to work together to solve everyday problems.
- *Social network expansion.* Therapists encourage people with schizophrenia and their families to expand their social contacts, especially their support networks.
- *Hope.* Therapists instil hope that things can improve, including the hope that the person with schizophrenia may not have to return to the hospital.

Therapists use various techniques to implement these strategies. Examples include identifying stressors that could cause relapse, training families in communication skills and problem solving, and having high-EE family members watch videos of interactions of low-EE families (Penn & Mueser, 1996). Compared with standard treatments (usually just medication), family therapy plus medication has typically lowered relapse over periods of one to two years. This positive finding is evident particularly in studies in which the treatment lasted for at least nine months (Falloon et al., 1982, 1985; Hogarty et al., 1986, 1991; Kopelowicz & Liberman, 1998; McFarlane et al., 1995; Penn & Mueser, 1996).

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Family therapy can help educate people with schizophrenia and their families about schizophrenia and reduce expressed emotion.



### **Cognitive-behavioural therapy**

At one time, researchers assumed that it was futile to try to alter the cognitive distortions, including delusions, of people with schizophrenia. Now, however, a growing body of evidence demonstrates that the maladaptive beliefs of some people with schizophrenia can in fact benefit from cognitive-behavioural therapy (CBT) (Garety, Fowler, & Kuipers, 2000; Wykes, Steel, Everitt, & Tarrier, 2008).

People with schizophrenia can be encouraged to test out their delusional beliefs in much the same way as people without schizophrenia do. Through collaborative discussions (and in the context of other modes of treatment, including antipsychotic drugs), some people with schizophrenia have been helped to attach a non-psychotic meaning to paranoid symptoms and thereby reduce their intensity and aversive nature, similar to what is done for depression and panic disorder (Beck & Rector, 2000; Drury, Birchwood, Cochrane, & Macmillan, 1996; Haddock, Tarrier, Spaulding, Yusupoff, & McCarthy, 1998). Researchers have found that CBT can also reduce negative symptoms, for example, by challenging belief structures tied to low expectations for success (avolition) and low expectations for pleasure (anticipatory pleasure deficit in anhedonia) (Grant, Huh, Perivoliotis, Stolar, & Beck, 2012; Wykes et al., 2008).

Results from meta-analyses of over 50 studies of more than 2000 people with schizophrenia across eight countries found small to moderate effect sizes for positive symptoms, negative symptoms, mood and general life functioning (Jauhar, McKenna, Radua, & Laws, 2014; Wykes et al., 2008). The results are less encouraging for negative symptoms, but CBT is currently the most effective treatment for these symptoms (Elis et al., 2013). CBT has been used as an adjunctive treatment for schizophrenia in Great Britain for over 10 years and the results have been positive, even in community settings (Sensky et al., 2000; Turkington, Kingdom, & Turner, 2002; Wykes et al., 2008) and for people with schizophrenia who refuse to take antipsychotic medications (Morrison et al., 2014).

Cognitive-behavioural therapy has also been used in the treatment of people meeting the at-risk mental state criteria. CBT provides a treatment that does not have the adverse effects of antipsychotic medication, helps break down the isolation of people in this situation and provides them with a cognitive framework with which to cope with their unusual perceptual experiences and ideas. It has reduced the transition rate to psychosis in some (Bechdolf et al., 2012; Morrison et al., 2004; van der Gaag et al., 2012), though not all, studies (Morrison et al., 2012).

### **Cognitive remediation therapies**

Researchers and clinicians have been paying more attention to fundamental aspects of cognition that are disordered in schizophrenia in an attempt to improve these functions and thereby favourably affect behaviour. This general approach concentrates on trying to normalise such functions as attention and memory, which are known to be deficient in many people with schizophrenia and are associated with poor social adaptation (Green, Kern, Braff, & Mintz, 2000).

Treatments that seek to enhance basic cognitive functions such as verbal learning ability are referred to as **cognitive remediation therapy** or *cognitive enhancement therapy (CET)* or *cognitive training*. A two-year randomised controlled clinical trial compared group-based CET with enriched supportive therapy (EST). CET consisted of nearly 80 hours of computer-based training in attention, memory and problem solving. Groups also worked on such routine social-cognitive skills as reading and understanding newspaper editorials, solving social problems and starting and maintaining conversations. EST included supportive and educational elements. All people were also taking medications. At the one- and two-year follow-up assessments, CET was more effective than EST in improving cognitive abilities in problem solving, attention, social cognition and social adjustment, while symptom reduction was the same for both treatments (Hogarty, Flesher, Ulrich et al., 2004). People who received CET were also rated as being more ready for employment and, in fact, tended to be employed at the end of two years, largely driven by the fact that these people were more likely to be in volunteer positions than those in the EST group. A different study compared CET with supportive therapy and found that CET was associated with improvements in social functioning that remained one year after the treatment ended (Eack, Greenwald, Hogarty, & Keshavan, 2010). Thus, CET appears to be effective in reducing symptoms and improving cognitive abilities and it is linked to good functional outcomes, such as employment and social functioning.

Two meta-analyses of 26 (McGurk et al., 2007) and 40 studies (Wykes, Huddy, Cellard et al., 2011) found small to medium effect sizes for overall cognitive functioning and specific cognitive domains, including attention, verbal memory, problem solving, verbal working memory, processing speed and social cognition. Cognitive remediation was also associated with a reduction in symptoms and an improvement in everyday functioning, though the effect sizes for these two domains were small. Cognitive remediation was more likely to be associated with an improvement in functioning if another type of psychosocial treatment, such as social skills training, was added to the treatment program. As promising as these findings are, their generalisability remains to be established.

A somewhat different cognitive remediation program has also shown promising results. This treatment also involves intensive (50 hours) computer training, but the task was developed based on neuroscience research showing that improving basic cognitive and perceptual processes (e.g., discriminating simple sounds) can then impact higher-order cognitive processes (e.g., memory and problem solving). The cognitive remediation task in this training is an auditory task that gets progressively more difficult as people get better at it. They perform a series of tasks that require them to discriminate between complex speech sounds. In a recent randomised controlled trial, people with schizophrenia who received the intensive auditory computer training showed greater improvement in overall cognition as well as in specific domains (memory, attention, processing speed, working memory and problem solving) compared to participants who played computer games for the same amount of time (Fisher, Holland, Merzenich, & Vinogradov, 2009).

Cognitive remediation therapy aimed specifically at social cognitive deficits has also been developed. This aims to treat the underlying dysfunction in social cognitive domains such as emotion recognition,

theory of mind deficits and attributional biases. These social cognitive deficits are a significant cause of disability, as significant as neurocognition (Fett, Viechtbauer, et al., 2011), and may mediate the effect of neurocognition on outcome (Schmidt, Mueller, & Roder, 2011). A range of treatments has been developed to address these deficits (Kurtz & Richardson, 2012), including programs that specifically teach individuals where to look to understand facial emotion (Marsh, Lockett, Russell, Coltheart, & Green, 2012) or help improve a person's understanding of the implicit social rules of an interaction or what other people might be thinking or feeling (Horan et al., 2011; Roberts & Penn, 2009). These treatments are still under development, however, they offer a different approach to the intractable levels of social disability experienced by people with schizophrenia.

## Psychoeducation

As we discussed in the chapter on mood disorders with regard to bipolar disorder, psychoeducation is an approach that seeks to educate people about their illness, including the symptoms of the disorder, the expected time course of symptoms, the biological and psychological triggers for symptoms and treatment strategies. A recent meta-analysis of 44 studies of psychoeducation in schizophrenia found that, in combination with medication, it was effective in reducing relapse and rehospitalisation and increasing medication compliance (Xia, Merinder, & Belgamwar, 2011).

## Case management

After large numbers of people were discharged from hospitals (referred to as deinstitutionalisation) in the 1960s, many people with schizophrenia no longer resided in hospitals and thus had to fend for themselves in securing needed services. Lacking the centralised hospital as the site where most services were delivered, the mental health system became more complex. To cope with this, community mental health services were established to help coordinate and provide services to people with a severe mental illness like schizophrenia. These services have been developed in a number of different models.

The simplest model is that of the case manager as the *broker* of services. Because case managers were familiar with the system, they were able to connect people with schizophrenia with providers of whatever services they required. Alternatively, an *Extended Hours Team* can be developed that provides case management and emergency care if a person relapses, but in a reactive way. Another model is that of *assertive case management*. In this model the case managers provided direct clinical services in a multidisciplinary team rather than broker them out. The assertive community treatment model (Stein & Test, 1980) and the intensive case management model (Surles, Blanch, Shern, & Donahue, 1992) both entail a multidisciplinary team that provides services in the community, such as medication, treatment for substance abuse, help in dealing with stressors that people with schizophrenia face regularly (such as managing money), psychotherapy, vocational training, and assistance in obtaining housing and employment. Case managers hold together and coordinate the range of medical and psychological services that people with schizophrenia need to keep functioning outside of institutions with some degree of independence and peace of mind (Kopelowicz et al., 2002). Both of these models continue to be part of the treatment guidelines for effective community care for people with schizophrenia (Dixon et al., 2010; Galletly et al., 2016; National Collaborating Centre for Mental Health, 2014).

This more intensive treatment has proven superior to less intensive methods in reducing time spent in the hospital, improving housing stability and ameliorating symptoms (Mueser, Bond, Drake, & Resnick, 1998). However, more intensive case management has not shown positive effects in other domains, such as improvement in social functioning. In order for this approach to be effective, there have to be enough case managers for people with schizophrenia. Too often, the caseloads of these mental health professionals are much too large and so they are spread too thin.

## Supported employment

An important issue for people with schizophrenia is their exclusion from the workforce. In Australia only 22.4 percent of people with psychosis are in full-time paid employment, despite this being one of their chief concerns (Waghorn et al., 2012). Worryingly, this has not changed in the 15 years between

national surveys. People with schizophrenia have many barriers to open employment; however, it is clear that a significant number of people with schizophrenia can return to work or education. Supported employment is a model of intervention that places people with a disability directly in the workplace and then supports them in maintaining that employment. The model is most successful when the employment services are integrated with the mental health services and the supporting employment specialist has full access to the individual in their workplace. Using the individual placement and support model, 61 percent of people with a severe mental illness returned to competitive employment compared to 23 percent of control participants (Bond, Drake, & Becker, 2008). This model has successfully returned young people back to education as well as employment (Killackey, Jackson, & McGorry, 2008) and is even more successful when combined with cognitive remediation training that helps address some of the cognitive deficits suffered by people with schizophrenia (Bell, Byrson, Greig, Corcoran, & Wexler, 2001; McGurk, Mueser, Feldman, Wolfe, & Pascaris, 2007; McGurk et al., 2015).

## **Recovery**

With the growth of the consumer movement in mental health, there has been a focus on different models of health that emphasise wellbeing and individual control rather than illness. These different models are based on a concept of recovery that might be best defined as:

... a deeply personal, unique process of changing one's attitudes, values, feelings, goals, skills and/or roles. It is a way of living a satisfying, hopeful and contributing life even with limitations caused by illness. Recovery involves the development of new meaning and purpose in one's life as one grows beyond the catastrophic effects of mental illness. (Anthony, 1993, p. 527)

As such, recovery has qualities that are outside the usual biomedical model of treatment in that it is an individual process that anticipates growth and development as part of a journey. It places the individual at the centre, empowering that person, giving them the right and responsibility to decide upon their own goals and make their own decisions. It sees the journey as one made with others as people become integrated into the community rather than being segregated, stigmatised because of their illness (Slade, 2009).

Recovery has become an important principle in the framing of policy in mental health (Department of Health and Ageing, 2013; Mental Health Commission, 2012; World Health Organization, 2013). This has helped with the introduction of a range of interventions such as mental health peer workers; however, the evidence base for recovery practice remains small.

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## SUMMARY

### **6.1 Describe the clinical symptoms of schizophrenia, including positive, negative and disorganised symptoms.**

Schizophrenia is a very heterogeneous disorder. It typically begins in late adolescence or early adulthood. Symptoms can be distinguished as positive, negative, disorganised, cognitive and mood/anxiety. Positive symptoms include hallucinations and delusions; negative symptoms include avolition, alogia, blunted affect, anhedonia and asociality. Together, the negative symptoms represent two domains: motivation/pleasure and expression. Disorganised symptoms include disorganised speech and disorganised behaviour. Cognitive symptoms include both neurocognitive (e.g., attention, memory, executive function and speed of processing) and social cognition (emotion recognition, theory of mind and attributional bias). People with schizophrenia also suffer from high levels of anxiety and depression. The DSM-5 no longer includes schizophrenia subtypes because they do not have adequate validity and are not very useful. Severity ratings for each of the schizophrenia symptoms were added in DSM-5. Other psychotic disorders include schizophreniform disorder and brief psychotic disorder, which differ from schizophrenia in duration. Schizoaffective disorder involves symptoms of both schizophrenia and either a depressive or manic episode. Delusional disorder involves delusions but no other symptoms of schizophrenia. The category attenuated psychosis syndrome, which involves positive symptoms in attenuated form that cause distress and have worsened in the past year, was not included in DSM-5 because the research to date is limited. Instead, it was added to the list of conditions in need of further study.

### **6.2 Differentiate the genetic factors, both behavioural and molecular, in the aetiology of schizophrenia.**

Given its complexity, a number of causal factors are likely to contribute to schizophrenia. Schizophrenia is a polygenetic disorder with a significant environmental input. The genetic evidence is strong, with much of it coming from family, twin and adoption studies. Familial high-risk studies have found that children with a biological parent with schizophrenia are more likely to develop adult psychopathology, including schizophrenia, and have difficulties with attention and motor control, among other things. Molecular genetics studies have identified both *de novo* and rare alleles, and copy number variations (CNVs) associated with schizophrenia that are usually rare causes of the disorder. Genome-wide association studies have identified a large number of genes of small effect associated with genetic vulnerability to schizophrenia. It is expected that more of these will be identified as the studies grow in size.

### **6.3 Describe how the brain has been implicated in schizophrenia.**

Neurotransmitters play a role in schizophrenia. For years, dopamine was the focus of study, but later findings led investigators to conclude that this one neurotransmitter could not fully account for schizophrenia. Other neurotransmitters are also the focus of study, such as serotonin, GABA and glutamate. Early neuroimaging identified changes in the neuroanatomy of the brain such as enlarged lateral ventricles. Although other research supports the role of the prefrontal cortex, particularly reduced activation of this area or temporal lobe changes, it is likely that the pathology is distributed and may be a property of a dysfunctional network between different brain areas in schizophrenia.

### **6.4 Describe the role of environmental factors in the aetiology and relapse of schizophrenia.**

Environmental factors, such as obstetric complications and prenatal infections, may impact the developing brain and increase the risk of schizophrenia. Cannabis use among adolescents has been associated with greater risk of schizophrenia, particularly among those who are genetically vulnerable to the disorder.

Research has examined the role of socioeconomic status in schizophrenia and generally this work supports the social selection hypothesis more than the sociogenic hypothesis. Early theories

blamed families, particularly mothers, for causing schizophrenia, but research does not support this view. Communication in families is important and could perhaps constitute the stress in the diathesis–stress theory of schizophrenia. Expressed emotion has also been found to predict relapse in schizophrenia, though there are important cultural differences in expressed emotion.

Retrospective developmental studies looked back at the childhood records of adults with schizophrenia and found that some adults with schizophrenia had lower IQs and were withdrawn and delinquent as children. Other studies found that adults who later developed schizophrenia expressed a lot of negative emotion and had poor motor skills. A prospective study confirmed that lower IQ in childhood is a predictor of the later onset of schizophrenia and that the IQ deficits are stable across childhood, though there is a later deterioration in cognition with illness. Clinical high-risk studies identify people who are showing early signs of schizophrenia.

#### **6.5 Discuss the appropriate use of medication and psychological treatments for schizophrenia.**

Antipsychotic medications remain the cornerstone of treatment for schizophrenia; however, their use is limited by significant adverse effects that lower their tolerability, and poor treatment adherence. All available effective antipsychotic medications are dopamine receptor blocking agents, however, the introduction of second-generation antipsychotics, particularly clozapine, has broadened the possible role of other neurotransmitters in the mode of action. Good treatment requires the integration of psychosocial interventions into the pharmacological management of schizophrenia. These psychosocial interventions include psychoeducation, social skills training, assertive case management, cognitive–behavioural therapy, cognitive remediation therapy and supported employment. Treatment should be provided in a recovery-focused framework that involves the person with schizophrenia at the centre of treatment decisions.

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## **KEY TERMS**

**alogia** a negative symptom in schizophrenia, marked by poverty of thought and speech

**anhedonia** a negative symptom in schizophrenia or a symptom in depression in which the individual experiences a loss of interest and pleasure

**anticipatory pleasure** expected or anticipated pleasure for events, people or activities in the future

**asociality** a negative symptom of schizophrenia marked by the loss of the importance of relationships, and an inability to form close relationships and to feel intimacy

**avolition** a negative symptom in schizophrenia in which the individual lacks interest and drive

**blunted affect** a negative symptom of schizophrenia that involves a lack of outward expression of emotion

**brief psychotic disorder** a disorder in which a person has a sudden onset of psychotic symptoms — incoherence, loose associations, delusions, hallucinations — immediately after a severely disturbing event; the symptoms last more than one day but no more than one month

**catatonia** constellation of schizophrenia symptoms including repetitive, peculiar, complex gestures and, in some cases, an almost manic increase in overall activity level. It can also manifest as immobility, with a fixity of posture maintained for long periods, with accompanying muscular rigidity, trancelike state of consciousness and waxy flexibility

**clinical high-risk study** a study that identifies people who show subtle or early clinical signs of a disorder, such as schizophrenia, and then follows them over time to determine who might be at risk for developing the disorder

**cognitive remediation therapy (CRT)** a behavioural training based intervention that targets cognitive processes such as attention, concentration, memory, executive function, speed of processing, social cognition or metacognition

**consummatory pleasure** pleasure experienced in the moment or in the presence of a pleasurable stimulus

**delusional disorder** a disorder in which the individual has persistent delusions and is very often contentious but has no disorganised thinking or hallucinations

**delusions** beliefs contrary to reality, firmly held in spite of evidence to the contrary and out of keeping with the person's own community or cultural background. They can include: of control, belief that one is being manipulated by some external force such as radar, television or a creature from outer space; of grandeur, belief that one is an especially important or powerful person; of persecution, belief that one is being plotted against or oppressed by others

**dementia praecox** an older term for schizophrenia, believed then to be an incurable and progressive deterioration of mental functioning beginning in adolescence

**derailment** see *loose associations*

**disorganised behaviour** symptom of schizophrenia that is marked by odd behaviours that do not appear organised, such as bouts of agitation, unusual dress or childlike, silly behaviour

**disorganised speech** speech found in schizophrenia patients that is marked by poorly organised ideas and speech that is difficult for others to understand; also known as formal thought disorder

**disorganised symptoms** broad category of symptoms in schizophrenia that includes disorganised speech, disorganised thinking and disorganised behaviour

**expressed emotion (EE)** hostility, criticism and emotional overinvolvement directed from other people towards the patient, usually within a family

**familial high-risk study** a study involving the offspring of people with a disorder, such as schizophrenia, who have a high probability of later developing a disorder

**grandiose delusion** a delusion of an exaggerated sense of one's importance, power, knowledge or identity

**hallucinations** perceptions in any sensory modality without relevant and adequate external stimuli

**ideas of reference** delusional thinking that reads personal significance into seemingly trivial remarks or activities of others and completely unrelated events

**loose associations (derailment)** in schizophrenia, an aspect of disorganised thinking wherein the patient has difficulty sticking to one topic and drifts off on a train of associations

**loss of insight** a loss of the ability of a person to recognise that their symptoms, such as hallucinations or delusions, are part of an illness, that they need treatment to help control these symptoms and that it is reasonable for them to see mental health professionals for this help

**negative symptoms** a symptom domain characterised by deficits that include blunted affect, anhedonia, asociality, alogia and avolition

**positive symptoms** a symptom domain characterised by hallucinations and delusions

**prefrontal cortex** the region of the frontal lobe of the brain that helps maintain an image of threats and rewards faced, as well as maintain focus and plans relevant to those threats and rewards

**schizoaffective disorder** diagnosis applied when a patient has symptoms of both mood disorder and either schizophreniform disorder or schizophrenia

**schizophrenia** a disorder characterised by disturbances in thought, emotion, behaviour, cognition and mood; disordered thinking in which ideas are not logically related; delusional beliefs; faulty perception, such as hallucinations; disturbances in attention; disturbances in motor activity; blunted expression of emotion; reduced desire for interpersonal relations and withdrawal from people; diminished motivation and anticipatory pleasure; cognitive deficits in attention, concentration, memory, executive functioning and social cognition

**schizophreniform disorder** diagnosis given to people who have all the symptoms of schizophrenia for more than four weeks but less than six months

**second-generation antipsychotic drugs** any of several drugs, such as clozapine, used to treat schizophrenia that produce fewer motor adverse effects than traditional antipsychotics while reducing positive and disorganised symptoms at least as effectively; may, however, be associated with increased and serious adverse effects of other varieties

**social selection hypothesis** an attempt to explain the correlation between social class and schizophrenia by arguing that people with schizophrenia tend to move downward in socioeconomic status

**social skills training** behaviour therapy procedures, such as modelling and behaviour rehearsal, for teaching individuals how to meet others, talk to them and maintain eye contact, give and receive criticism, offer and accept compliments, make requests and express feelings and otherwise improve their relations with other people

**sociogenic hypothesis** an idea that seeks causes in social conditions, for example, that being in a low social class can cause one to develop schizophrenia

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## WEBSITES

1. *Schizophrenia Bulletin* is the leading specialist journal in the area of schizophrenia, with much of its content open to the public. It provides high-quality scientific articles on all aspects of schizophrenia, and includes special sections devoted to first-person accounts of living with schizophrenia and on evidence-based treatment. (<https://academic.oup.com/schizophreniabulletin>)
2. Schizophrenia Research Forum is an open website dedicated to fostering the best research possible into schizophrenia, offering high-quality information about schizophrenia and a meeting place for scientists and the public. ([www.schizophreniaforum.org](http://www.schizophreniaforum.org))
3. SANE is a non-government organisation that hosts a number of forums throughout Australia and online exploring schizophrenia and other mental illnesses; provides good quality consumer-oriented information. ([www.sane.org](http://www.sane.org))
4. Mental Health Foundation of New Zealand has a plethora of resources and personal stories that provide a broad understanding of the effect of schizophrenia and other mental illnesses on individuals and what communities can do about it. ([www.mentalhealth.org.nz](http://www.mentalhealth.org.nz))

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## REFERENCES

- Addington, D., Addington, J., & Patten, S. (1998). Depression in people with first-episode schizophrenia. *British Journal of Psychiatry*, 172 (suppl.33), 90–92.
- Albee, G. W., Lane, E. A., & Reuter, J. M. (1964). Childhood intelligence of future schizophrenics and neighborhood peers. *Journal of Psychology*, 58, 141–144.
- Allen, P., Johns, L. C., Fu, C. H. Y., Broome, M. R., Vythelingum, G. N., & McGuire, P. K. (2004). Misattribution of external speech in patients with hallucinations and delusions. *Schizophrenia Research*, 69, 277–287.
- Amador, X. F., Flaum, M., Andreasen, N. C., Strauss, D. H., Yale, S. A., et al. (1994). Awareness of illness in schizophrenia and schizoaffective and mood disorder. *Archives of General Psychiatry*, 51, 826–836.
- Andreasen, N. (1979). Thought, Language and Communication Disorders. 1. Clinical assessment, definition of terms and evaluation of their reliability. *Archives of General Psychiatry*, 36, 1315–1321.
- Andreasen, N. C., Liu, D., Ziebell, S., Vora, A., & Ho, B. C. (2013). Relapse duration, treatment intensity, and brain tissue loss in schizophrenia: A prospective longitudinal MRI Study. *American Journal of Psychiatry*, 170(6), 609–615. doi: 10.1176/appi.ajp.2013.12050674
- Andreasen, N. C., Olsen, S. A., Dennert, J. W., & Smith, M. R. (1982). Ventricular enlargement in schizophrenia: Relationship to positive and negative symptoms. *American Journal of Psychiatry*, 139, 297–302.
- Angrist, B., Lee, H. K., & Gershon, S. (1974). The antagonism of amphetamine-induced symptomatology by a neuroleptic. *American Journal of Psychiatry*, 131, 817–819.
- Anthony, W. A. (1993). Recovery from mental illness: The guiding vision of the mental health system in the 1990s. *Psychosocial Rehabilitation Journal*, 16(4), 11–23.
- Arnedo, J., Svrakic, D. M., Val, C. D., Romero-Zalaz, R., Hernández-Cuervo, H., Consortium, M. G. O. S., ... Zwir, I. (2015). Uncovering the hidden risk architecture of the schizophrenias: Confirmation in three independent genome-wide association studies. *American Journal of Psychiatry*, 172(2), 139–153. doi:10.1176/appi.ajp.2014.14040435
- Arseneault, L., Cannon, M., Poulton, R., Murray, R., Caspi, A., & Moffitt, T. E. (2002). Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. *British Medical Journal*, 325, 1212–1213.
- Barch, D. M., Carter, C. S., Braver, T. S., et al. (2001). Selective deficits in prefrontal cortex function in medication naïve patients with schizophrenia. *Archives of General Psychiatry*, 58, 280–288.

- Barch, D. M., Carter, C. S., MacDonald, A. W., Braver, T. S., & Cohen, J. D. (2003). Context processing deficits in schizophrenia: Diagnostic specificity, four-week course, and relationship to clinical symptoms. *Journal of Abnormal Psychology, 112*, 132–143.
- Barch, D. M., Csernansky, J. G., Conturo, T., & Snyder, A. Z. (2002). Working and long-term memory deficits in schizophrenia: Is there a common prefrontal mechanism? *Journal of Abnormal Psychology, 111*, 478–494.
- Baumeister, A. A., & Francis, J. L. (2002). Historical development of the dopamine hypothesis of schizophrenia. *Journal of the History of the Neurosciences, 11*(3), 265–277.
- Bechdolf, A., Nelson, B., Cotton, S. M., Chanen, A., Thompson, A., Kettle, J., . . . McGorry, P. D. (2010). A preliminary evaluation of the validity of at-risk criteria for bipolar disorders in help-seeking adolescents and young adults. *Journal of Affective Disorders, 127*(1), 316–320. doi: 10.1016/j.jad.2010.06.016
- Bechdolf, A., Wagner, M., Ruhrmann, S., Harrigan, S., Putzfeld, V., Pukrop, R., . . . Klosterkötter, J. (2012). Preventing progression to first-episode psychosis in early initial prodromal states. *British Journal of Psychiatry, 200*, 22–29. doi: 10.1192/bjp.bp.109.066357
- Beck, A. T., & Rector, N. A. (2000). Cognitive therapy of schizophrenia: A new therapy for the new millennium. *American Journal of Psychotherapy, 54*, 291–300.
- Bell, M., Byrson, G., Greig, T., Corcoran, C., & Wexler, B. E. (2001). Neurocognitive Enhancement Therapy with work therapy. *Archives of General Psychiatry, 58*, 763–768.
- Berry, J. C. (1967). *Antecedents of schizophrenia, impulsive character and alcoholism in males*. Paper presented at the 75th Annual Convention of the American Psychological Association, Washington, DC.
- Blanchard, J. J., & Cohen, A. S. (2006). The structure of negative symptoms within schizophrenia: implications for assessment. *Schizophrenia Bulletin, 32*, 238–245.
- Blanchard, J. J., Squires, D., Henry, T., Horan, W. P., Bogenschutz, M., et al. (1999). Examining an affect regulation model of substance abuse in schizophrenia: The role of traits and coping. *Journal of Nervous and Mental Disease, 187*, 72–79.
- Bond, G. R., Drake, R. E., & Becker, D. R. (2008). An update on randomised controlled trials of evidence-based supported employment. *Psychiatric Rehabilitation Journal, 31*(4), 280–290.
- Boos, H. B., Aleman, A., Cahn, W., Hulshoff, H., & Kahn, R. S. (2007). Brain volumes in relatives of patients with schizophrenia: A meta-analysis. *Archives of General Psychiatry, 64*, 297–304.
- Bora, E., Fornito, A., Radua, J., Walterfang, M., Seal, M., Wood, S. J., . . . Pantelis, C. (2011). Neuroanatomical abnormalities in schizophrenia: A multimodal voxelwise meta-analysis and meta-regression analysis. *Schizophrenia Research, 127*(1–3), 46–57.
- Braff, D. L., Ryan, J., Rissling, A. J., & Carpenter, W. T. (2013). Lack of use in the literature from the last 20 years supports dropping traditional schizophrenia subtypes from DSM-5 and ICD-11. *Schizophrenia Bulletin, 39*(4), 751–753.
- Brown, A. S. (2011). The environment and susceptibility to schizophrenia. *Progress in Neurobiology, 93*, 23–58.
- Brown, A. S., & Derkits, E. J. (2010). Prenatal infections and schizophrenia: A review of epidemiologic and translational Studies. *American Journal of Psychiatry, 167*, 261–280.
- Brown, A. S., Begg, M. D., Gravenstein, S., Schaefer, C. A., Wyatt, R. J., Bresnahan, M., Babulas, V. P., & Susser, E. S. (2004). Serologic evidence of prenatal influenza in the etiology of schizophrenia. *Archives of General Psychiatry, 61*, 774–780.
- Brown, A. S., Bottiglieri, T., Schaefer, C. A., Quesenberry, C. P., Jr., Liu, L., Bresnahan, M., & Susser, E. S. (2007). Elevated prenatal homocysteine levels as a risk factor for schizophrenia. *Archives of General Psychiatry, 64*, 31–39.
- Brown, A. S., Schaefer, C. A., Quesenberry, C. P., Jr., Liu, L., Babulas, V. P., & Susser, E. S. (2005). Maternal exposure to toxoplasmosis and risk of schizophrenia in adult offspring. *American Journal of Psychiatry, 162*, 767–773.
- Brown, G. W., Bone, M., Dalison, B., & Wing, J. K. (1966). *Schizophrenia and social care*. London: Oxford University Press.
- Buchsbaum, M. S., Kessler, R., King, A., Johnson, J., & Cappelletti, J. (1984). Simultaneous cerebral glucography with positron emission tomography and topographic electroencephalography. In G. Pfurtscheller, E. J. Jonkman & F. H. L. d. Silva (Eds.), *Brain ischemia: Quantitative EEG and imaging techniques*. Amsterdam: Elsevier.
- Buckley, P. F., Miller, B. J., Lehrer, D. S., & Castle, D. J. (2009). Psychiatric comorbidities and schizophrenia. *Schizophrenia Bulletin, 35*, 383–402.
- Bulik-Sullivan, B., Finucane, H. K., Anttila, V., Gusev, A., Day, F. R., Loh, P.-R., . . . Neale, B. M. (2015). An atlas of genetic correlations across human diseases and traits. *Nat Genet, 47*(11), 1236–1241. doi: 10.1038/ng.3406 <http://www.nature.com/ng/journal/v47/n11/abs/ng.3406.html> - supplementary-information
- Burris, K. D., Molski T. F., Xu, C., Ryan, E., Tottori, K., Kikuchi, T., Yocca F. D., & Molinoff, P. B. (2002). Aripiprazole, a novel antipsychotic, is a high-affinity partial agonist at human dopamine D2 receptors. *Journal of Pharmacology and Experimental Therapeutics, 302*, 381–389.
- Butzlaff, R. L., & Hooley, J. M. (1998). Expressed emotion and psychiatric relapse: A meta-analysis. *Archives of General Psychiatry, 55*, 547–553.
- Cahalan, S. (2012). *Brain on fire: My month of madness*. New York: Free Press.
- Cannon, T. D., & Mednick, S. A. (1993). The schizophrenia high-risk project in Copenhagen: Three decades of progress. *Acta Psychiatrica Scandinavica, 87*, 33–47.
- Cannon, T. D., Kaprio, J., Lonnqvist, J., Huttunen, M., & Koskenvuo, M. (1998). The genetic epidemiology of schizophrenia in a Finnish twin cohort: A population-based modeling study. *Archives of General Psychiatry, 55*, 67–74.

- Cannon, T. D., van Erp, T. G., Rosso, I. M., et al. (2002). Fetal hypoxia and structural brain abnormalities in schizophrenic patients, their siblings, and controls. *Archives of General Psychiatry*, 59, 35–42.
- Cardno, A. G., Marshall, E. J., Coid, B., Macdonald, A. M., Ribchester, T. R., et al. (1999). Heritability estimates for psychotic disorders: The Maudsley Twin Psychosis Series. *Archives of General Psychiatry*, 56, 162–170.
- Carlsson, A., Hanson, L. O., Waters, N., & Carlsson, M. L. (1999). A glutamatergic deficiency model of schizophrenia. *British Journal of Psychiatry*, 174, 2–6.
- Carpenter, W. T., & van Os, J. (2011). Should attenuated psychosis syndrome be a DSM-5 diagnosis? *American Journal of Psychiatry*, 168, 460–463.
- Caspi, A., Moffitt, T. E., Cannon, M., McClay, J., Murray, R., Harrington, H., Taylor, A., Arseneault, L., Williams, B., Braithwaite, A., Pulton, R., & Craig, I. W. (2005). Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-O-methyltransferase gene: Longitudinal evidence of a gene–environment interaction. *Biological Psychiatry*, 57, 1117–1127.
- Chesney, E., Goodwin, G. M., & Fazel, S. (2014). Risks of all-cause and suicide mortality in mental disorders: A meta review. *World Psychiatry*, 13, 153–160.
- Collin, G., Kahn, R. S., de Reus, M. A., Cahn, W., & van den Heuvel, M. P. (2014). Impaired rich club connectivity in unaffected siblings of schizophrenia patients. *Schizophrenia Bulletin*, 40(2), 438–448.
- Copolov, D. L., Mackinnon, A., & Trauer, T. (2004). Correlates of the affective impact of auditory hallucinations in psychotic disorders. *Schizophrenia Bulletin*, 30, 163–171.
- Couture, S. M., Penn, D. L., & Roberts, D. L. (2006). The functional significance of social cognition in schizophrenia: A review. *Schizophrenia Bulletin*, 32, S44–S63.
- Crossley, N. A., Mechelli, A., Scott, J., Carletti, F., Fox, P. T., McGuire, P., & Bullmore, E. T. (2014). The hubs of the human connectome are generally implicated in the anatomy of brain disorders. *Brain*, 137(8), 2382–2395. doi: 10.1093/brain/awu132
- Curcio-Blake, B., Liemburg, E., Vercammen, A., Swart, M., Kneegtering, H., Bruggeman, R., & Aleman, A. (2013). When Broca goes uninformed: reduced information flow to Broca's area in schizophrenia patients with auditory hallucinations. *Schizophrenia Bulletin*, 39(5), 1087–1095.
- Dalmau, J., Tüzün, E., Wu, H. Y., Masjuan, J., Rossi, J. E., Voloschin, A., et al. (2007). Paraneoplastic anti-N-methyl-D-aspartate receptor encephalitis associated with ovarian teratoma. *Annals of Neurology*, 61(1), 25–36.
- Das, P., Kemp, A. H., Flynn, G., Harris, A. W. F., Liddell, B. J., Whitford, T. J., . . . Williams, L. M. (2007). Functional disconnections in the direct and indirect amygdala pathways for fear processing in schizophrenia. *Schizophrenia Research*, 90(1–3), 284–294.
- Davis A. S., Malmberg, A., Brandt, L., Allebeck, P., & Lewis, G. (1997). IQ and risk for schizophrenia: A population-based cohort study. *Psychological Medicine*, 27, 1311–1323.
- Deakin, J., Lennox, B. R., & Zandi, M. S. (2014). Antibodies to the N-methyl-D-aspartate receptor and other synaptic proteins in psychosis. *Biological Psychiatry*, 75(4), 284–291. doi: <http://dx.doi.org/10.1016/j.biopsych.2013.07.018>
- Department of Health and Ageing. (2013). *A national framework for recovery-oriented mental health services*. Canberra: Commonwealth of Australia.
- Deserno, L., Sterzer, P., Wustenberg, T., Heinz, A., & Schlagenhauf, F. (2012). Reduced prefrontal-parietal effective connectivity and working memory deficits in schizophrenia. *Journal of Neuroscience*, 32(1), 12–20.
- Dhindsa, R. S., & Goldstein, D. B. (2016). Schizophrenia: From genetics to physiology at last. *Nature*, 530(7589), 162–163. doi: 10.1038/nature16874
- Di Forti, M., Iyegbe, C., Sallis, H., Kolliakou, A., Falcone, M. A., Paparelli, A., . . . Murray, R. M. (2012). Confirmation that the AKT1 (rs2494732) genotype influences the risk of psychosis in cannabis users. *Biological Psychiatry*, 72, 811–816.
- Di Forti, M., Sallis H., Allegri, F., Trotta, A., Ferraro L., Stilo, S. A., et al. (2013). Daily use, especially of high-potency cannabis, drives the earlier onset of psychosis in cannabis users. *Schizophrenia Bulletin*, doi: 10.1093/schbul/sbt181
- Dixon, L. B., Dickerson, F., Bellack, A. S., Bennett, M., Dickinson, D., Goldberg, R. W., et al. (2010). The 2009 schizophrenia PORT psychosocial treatment recommendations and summary statements. *Schizophrenia Bulletin*, 36, 48–70.
- Dohrenwend, B. P., Levav, P. E., Schwartz, S., Naveh, G., Link, B. G., Skodol, A. E., & Stueve, A. (1992). Socioeconomic status and psychiatric disorders: The causation–selection issue. *Science*, 255, 946–952.
- Drake, R., Whitaker, A., Gates, C., & Cotton, P. (1985). Suicide among schizophrenics: A review. *Comprehensive Psychiatry*, 26(1), 90–100. doi: 10.1016/0010-440x(85)90053-7
- Drury, V., Birchwood, M., Cochrane, R., & Macmillan, R. (1996). Cognitive therapy and recovery from acute psychosis: A controlled trial. *British Journal of Psychiatry*, 169, 593–601.
- Dworkin, R. H., & Lenzenwenger, M. F. (1984). Symptoms and the genetics of schizophrenia: Implications for diagnosis. *American Journal of Psychiatry*, 141, 1541–1546.
- Dworkin, R. H., Lenzenwenger, M. F., & Moldin, S. O. (1987). Genetics and the phenomenology of schizophrenia. In P. D. Harvey & E. F. Walker (Eds.), *Positive and negative symptoms of psychosis*. Hillsdale, NJ: Lawrence Erlbaum.
- Eack, S. M., Greenwald, D. P., Hogarty, S. S., & Keshavan, M. S. (2010). One-year durability of the effects of cognitive enhancement therapy on functional outcome in early schizophrenia. *Schizophrenia Research*, 120, 210–216.
- Elis, O., Caponigro, J. M., & Kring, A. M. (2013). Psychosocial treatments for negative symptoms in schizophrenia: Current practices and future directions. *Clinical Psychology Review*, 33(8), 914–928.

- Elkis, H., Friedman, L., Wise, A., & Meltzer, H. T. (1995). Meta-analysis of studies of ventricular enlargement and cortical sulcal prominence in mood disorders. *Archives of General Psychiatry*, 52, 735–746.
- Ellison-Wright, I., & Bullmore, E. (2009). Meta-analysis of diffusion tensor imaging studies in schizophrenia. *Schizophrenia Research*, 108(1–3), 3–10.
- Falloon, I. R. H., Boyd, J. L., McGill, C. W., Razani, J., Moss, H. B., & Gilderman, A. N. (1982). Family management in the prevention of exacerbation of schizophrenia: A controlled study. *New England Journal of Medicine*, 306, 1437–1440.
- Falloon, I. H., Boyd, J. L., McGill, C. W., & et al. (1985). Family management in the prevention of morbidity of schizophrenia: Clinical outcome of a two-year longitudinal study. *Archives of General Psychiatry*, 42(9), 887–896. doi: 10.1001/archpsyc.1985.01790320059008
- Faustman, W. O., Bardgett, M., Faull, K. F., Pfefferman, A., & Cseransky, J. G. (1999). Cerebrospinal fluid glutamate inversely correlates with positive symptom severity in unmedicated male schizophrenic/schizoaffective patients. *Biological Psychiatry*, 45, 68–75.
- Fett, A.-K. J., Maat, A., & Investigators, G. (2011). Social cognitive impairments and psychotic symptoms: What is the nature of their association? *Schizophrenia Bulletin*. doi: 10.1093/schbul/sbr058
- Fett, A.-K. J., Viechtbauer, W., Dominguez, M.-d.-G., Penn, D. L., van Os, J., & Krabbendam, L. (2011). The relationship between neurocognition and social cognition with functional outcomes in schizophrenia: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, 35(3), 573–588.
- Fisher, M., Holland, C., Merzenich, M. M., & Vinogradov, S. (2009). Using neuroplasticity-based auditory training to improve verbal memory in schizophrenia. *American Journal of Psychiatry*, 166, 805–811.
- Fisher, M. (1971). Psychoses in the offspring of schizophrenic monozygotic twins and their normal cotwins. *British Journal of Psychiatry*, 118, 43–52.
- Fornito, A., Yoon, J., Zalesky, A., Bullmore, E. T., & Carter, C. S. (2011). General and specific functional connectivity disturbances in first-episode schizophrenia during cognitive control performance. *Biological Psychiatry*, 70(1), 64–72. doi: http://dx.doi.org/10.1016/j.biopsych.2011.02.019
- Fornito, A., Zalesky, A., & Breakspear, M. (2015). The connectomics of brain disorders. *Nature Reviews Neuroscience*, 16(3), 159–172. doi: 10.1038/nrn3901
- Friston, K. J. (1994). Functional and effective connectivity in neuroimaging: A synthesis. *Human Brain Mapping*, 2, 56–78.
- Friston, K., Brown, H. R., Siemerkus, J., & Stephan, K. E. (2016). The dysconnection hypothesis (2016). *Schizophrenia Research*, 176(2), 83–94. doi: 10.1016/j.schres.2016.07.014
- Fromer, M., Pocklington, A. J., Kavanagh, D. H., Williams, H. J., Dwyer, S., Gormley, P., ... O'Donovan, M. C. (2014). De novo mutations in schizophrenia implicate synaptic networks. *Nature*, 506, 179–184. doi:10.1038/nature12929
- Fromm-Reichmann, F. (1948). Notes on the development of treatment of schizophrenics by psychoanalytic psychotherapy. *Psychiatry*, 11, 263–273.
- Fusar-Poli, P., Bechdolf, A., Taylor, M. J., Bonoldi, I., Carpenter, W. T., Yung, A. R., & McGuire, P. (2012). At risk for schizophrenic or affective psychoses? A meta-analysis of DSM/ICD diagnostic outcomes in individuals at high clinical risk. *Schizophrenia Bulletin*. doi: 10.1093/schbul/sbs060
- Fusar-Poli, P., Bonoldi, I., Yung, A. R., Borgwardt, S., Kempton, M. J., Valmaggia, L., ... McGuire, P. (2012). Predicting psychosis: Meta-analysis of transition outcomes in individuals at high clinical risk. *Archives of General Psychiatry*, 69(3), 220–229. doi: 10.1001/archgenpsychiatry.2011.1472
- Galletly, C. A., Foley, D. L., Waterreus, A., Watts, G. F., Castle, D. J., McGrath, J. J., ... Morgan, V. A. (2012). Cardiometabolic risk factors in people with psychotic disorders: The second Australian national survey of psychosis. *Australian & New Zealand Journal of Psychiatry*, 46(8), 753–761. doi: 10.1177/0004867412453089
- Galletly, C., Castle, D., Dark, F., Humberstone, V., Jablensky, A., Killackey, E., ... Tran, N. (2016). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the management of schizophrenia and related disorders. *Australian and New Zealand Journal of Psychiatry*, 50(5), 410–472. doi: 10.1177/0004867416641195
- Gard, D. E., Kring, A. M., Germans Gard, M., Horan, W. P., & Green, M. F. (2007). Anhedonia in schizophrenia: Distinctions between anticipatory and consummatory pleasure. *Schizophrenia Research*, 93, 253–260.
- Gard, D. E., Sanchez, A. H., Starr, J., Cooper, S., Fisher, M., Rowlands, A., & Vinogradov, S. (2014). Using self-determination theory to understand motivation deficits in schizophrenia: The “why” of motivated behavior. *Schizophrenia Research*, 156(2–3), 217–222.
- Garety, P. A., Fowler, D., & Kuipers, E. (2000). Cognitive behavioral therapy for medication-resistant symptoms. *Schizophrenia Bulletin*, 26, 73–86.
- Glantz, L. A., & Lewis, D. A. (2000). Decreased dendritic spine density on prefrontal cortical pyramidal neurons in schizophrenia. *Archives of General Psychiatry*, 57, 65–73.
- Glausier, J. R., & Lewis, D. A. (2013). Dendritic spine pathology in schizophrenia. *Neuroscience*, 251(0), 90–107.
- Goldman-Rakic, P. S., & Selemon, L. D. (1997). Functional and anatomical aspects of prefrontal pathology in schizophrenia. *Schizophrenia Bulletin*, 23, 437–458.
- Gouzoulis-Mayfrank, E., Heekeren, K., Neukirch, A., Stoll, M., Stock, K., Obradovic, M., & Kovar, K. A. (2005). Psychological effects of (s)-Ketamine and N,N-Dimethyltryptamine (DMT): A double-blind cross-over study in healthy volunteers. *Pharmacopsychiatry*, 38, 301–311.
- Gottesman, I. I., McGuffin, P., & Farmer, A. E. (1987). Clinical genetics as clues to the “real” genetics of schizophrenia. *Schizophrenia Bulletin*, 13, 23–47.

- Grant, P. M., Huh, G., Perivoliotis, D., Stolar, N., & Beck, A. T. (2012). Randomized trial to evaluate the efficacy of cognitive therapy for low-functioning patients with schizophrenia. *Archives of General Psychiatry*, 69(121–127).
- Green, M. F., Kern, R. S., & Heaton, R. K. (2004). Longitudinal studies of cognition and functional outcome in schizophrenia: Implications for MATRICS. *Schizophrenia Research*, 72, 41–51.
- Green, M. F., Kern, R. S., Braff, D. L., & Mintz, J. (2000). Neurocognitive deficits and functional outcome in schizophrenia: Are we measuring the “right stuff”? *Schizophrenia Bulletin*, 26, 119–136.
- Green, M. F., Marshall, B. D., Wirshing, W. C., Ames, D., Marder, S. R., McGurk, S., Kern, R. S., & Mintz, J. (1997). Does risperidone improve verbal working memory in treatment-resistant schizophrenia? *American Journal of Psychiatry*, 154, 799–804.
- Guo, X., Zhai, J., Liu, Z., Fang, M., Wang, B., Wang, C., Hu, B., Sun, X., et al. (2010). Effect of antipsychotic medication alone vs combined with psychosocial intervention on outcomes of early-stage schizophrenia. *Archives of General Psychiatry*, 67, 895–904.
- Gur, R. E., Turetsky, B. I., Cowell, P. E., et al. (2000). Temporolimbic volume reductions in schizophrenia. *Archives of General Psychiatry*, 57, 769–776.
- Haddock, G., Tarrier, N., Spaulding, W., Yusupoff, L. K., & McCarthy, E. (1998). Individual cognitive-behavior therapy in the treatment of hallucinations and delusions: A review. *Clinical Psychology Review*, 18, 821–838.
- Hajima, S. V., Van Haren, N., Cahn, W., Koolschijn, P. C., Hulshoff Pol, H. E., & Kahn, R. S. (2013). Brain volumes in schizophrenia: a meta-analysis in over 18 000 subjects. *Schizophrenia Bulletin*, 39(5), 1129–1138.
- Heinrichs, R. W., & Zakzanis, K. K. (1998). Neurocognitive deficit in schizophrenia: a quantitative review of the evidence. *Neuropsychology*, 12, 426–445.
- Heinssen, R. K., Liberman, R. P., & Kopelowicz, A. (2000). Psychosocial skills training for schizophrenia: Lessons from the laboratory. *Schizophrenia Bulletin*, 26, 21–46.
- Higashi, K., Medic, G., Littlewood, K. J., Diez, T., Granström, O., & De Hert, M. (2013). Medication adherence in schizophrenia: factors influencing adherence and consequences of nonadherence, a systematic literature review. *Therapeutic Advances in Psychopharmacology*, 3(4), 200–218. doi: 10.1177/2045125312474019
- Ho, B. C., Nopoulos, P., Flaum, M., Arndt, S., & Andreasen, N. C. (1998). Two-year outcome in first-episode schizophrenia: Predictive value of symptoms for quality of life. *American Journal of Psychiatry*, 155, 1196–1201.
- Hogarty, G. E., Anderson, C. M., Reiss, D. J., Kornblith, S. J., Greenwald, D. P., et al. (1986). Family psychoeducation, social skills training, and maintenance chemotherapy in the aftercare treatment of schizophrenia: 1. One-year effects of a controlled study on relapse and expressed emotion. *Archives of General Psychiatry*, 43, 633–642.
- Hogarty, G. E., Anderson, C. M., Reiss, D. J., Kornblith, S. J., Greenwald, D. P., Ulrich, R. F., Carter, M., et al. The Environmental-Personal Indicators in the Course of Schizophrenia (EPICS) Research Group. (1991). Family psychoeducation, social skills training, and maintenance chemotherapy in the aftercare treatment of schizophrenia. *Archives of General Psychiatry*, 48, 340–347.
- Hogarty, G. E., Flesher, S., Ulrich, R., et al. (2004). Cognitive enhancement therapy for schizophrenia: Effects of a 2-year randomized trial on cognition and behavior. *Archives of General Psychiatry*, 61, 866–876.
- Hollingshead, A. B., & Redlich, F. C. (1958). *Social class and mental illness: A community study*. New York: John Wiley & Sons.
- Horan, W. P., Kern, R. S., Tripp, C., Helleman, G., Wynn, J. K., Bell, M., . . . Green, M. F. (2011). Efficacy and specificity of Social Cognitive Skills Training for outpatients with psychotic disorders. *Journal of Psychiatric Research*, 45(8), 1113–1122.
- Horan, W. P., Kring, A. M., & Blanchard, J. J. (2006). Anhedonia in schizophrenia: A review of assessment strategies. *Schizophrenia Bulletin*, 32, 259–273.
- Howes, O. D., Kambeitz, J., Kim, E., Stahl, D., Slifstein, M., Abi-Dargham, A., & Kapur, S. (2012). The nature of dopamine dysfunction in schizophrenia and what this means for treatment: Meta-analysis of imaging studies. *Archives of General Psychiatry*, doi: 10.1001/archgenpsychiatry.2012.169
- Howes, O. D., McCutcheon, R., Owen, M. J., & Murray, R. M. (2017). The role of genes, stress and dopamine in the development of schizophrenia. *Biological Psychiatry*, 81(1), 9–20. doi: <http://dx.doi.org/10.1016/j.biopsych.2016.07.014>
- Jardri, R., Pouchet, A., Pins, D., & Thomas, P. (2011). Cortical activation during auditory verbal hallucinations in schizophrenia: a coordinate-based meta-analysis. *American Journal of Psychiatry*, 168, 73–81.
- Jauhar, S., McKenna, P. J., Radua, J., Fung, E., Salvador, R., & Laws, K. R. (2014). Cognitive-behavioural therapy for the symptoms of schizophrenia: Systematic review and meta-analysis with examination of potential bias. *British Journal of Psychiatry*, 204, 20–29.
- Jepson, J. (2016). A vet's recovery. *Schizophrenia Bulletin*, 42(1), 4. doi: 10.1093/schbul/sbv115
- Kane, J. M., Honigfeld, G., Singer, J., Meltzer, H. Y., & Clozaril Collaborative Study, G. (1988). Clozapine for the treatment-resistant schizophrenic: a double blind comparison with chlorpromazine. *Archives of General Psychiatry*, 45, 789–796.
- Kempton, M. J., Stahl, D., Williams, S. C., & DeLisi, L. E. (2010). Progressive lateral ventricular enlargement in schizophrenia: A meta-analysis of longitudinal MRI studies. *Schizophrenia Research*, 120, 54–62.
- Kerns, J. G., & Berenbaum, H. (2002). Cognitive impairments associated with formal thought disorder in people with schizophrenia. *Journal of Abnormal Psychology*, 111, 211–224.
- Kerns, J. G., & Berenbaum, H. (2003). The relationship between formal thought disorder and executive functioning component processes. *Journal of Abnormal Psychology*, 112, 339–352.
- Keshavan, M. S., Rosenberg, D., Sweeney, J. A., & Pettegrew, J. W. (1998). Decreased caudate volume in neuroleptic-naive psychotic patients. *American Journal of Psychiatry*, 155, 774–778.

- Killackey, E., Jackson, H. J., & McGorry, P. D. (2008). Vocational intervention in First-Episode Psychosis: A randomised controlled trial of individual placement and support versus treatment as usual. *British Journal of Psychiatry*, 193(2), 114–120.
- Kirkpatrick, B., Fenton, W., Carpenter, W. T., & Marder, S. R. (2006). The NIMH-MATRICES consensus statement on negative symptoms. *Schizophrenia Bulletin*, 32, 296–303.
- Kishimoto, T., Agarwal, V., Kishi, T., Leucht, S., Kane, J. M., & Correll, C. U. (2013). Relapse prevention in schizophrenia: a systematic review and meta-analysis of second-generation antipsychotics versus first-generation antipsychotics. *Molecular Psychiatry*, 18(1), 53–66. doi: <http://www.nature.com/mp/journal/v18/n1/supinfo/mp2011143s1.html>
- Kohn, M. L. (1968). Social class and schizophrenia: A critical review. In D. Rosenthal & S. S. Kety (Eds.), *The transmission of schizophrenia*. Elmsford, NY: Pergamon Press.
- Kopelowicz, A., & Liberman, R. P. (1998). Psychosocial treatments for schizophrenia. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 190–211). New York: Oxford University Press.
- Kopelowicz, A., Liberman, R. P., & Zarate, R. (2002). Psychosocial treatments for schizophrenia. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that Work*, 2nd Edition (pp. 201–229). New York: Oxford University Press.
- Kreyenbuhl, J., Buchanan, R. W., Dickerson, F. B., & Dixon, L. B. (2010). The schizophrenia patient outcomes research team (PORT): Updated treatment recommendations 2009. *Schizophrenia Bulletin*, 36, 94–103.
- Kring, A. M. (1999). Emotion in schizophrenia: Old mystery, new understanding. *Current Directions in Psychological Science*, 8, 160–163.
- Kring, A. M., & Caponigro, J. M. (2010). Emotion in schizophrenia: Where feeling meets thinking. *Current Directions in Psychological Science*, 19, 255–259.
- Kring, A. M., & Moran, E. K. (2008). Emotional response deficits in schizophrenia: Insights from affective science. *Schizophrenia Bulletin*, 38, 819–834.
- Kring, A. M., Gur, R. E., Blanchard, J. J., Horan, W. P., & Reise, S. P. (2013). The Clinical Assessment Interview for Negative Symptoms (CAINS): Final development and validation. *American Journal of Psychiatry*, 170(2), 165–172.
- Kurtz, M. M., & Richardson, C. L. (2012). Social cognitive training for schizophrenia: A meta-analytic investigation of controlled research. *Schizophrenia Bulletin*, 38(5), 1092–1104. doi:10.1093/schbul/sbr036
- Lane, E. A., & Albee, G. W. (1965). Childhood intellectual differences between schizophrenic adults and their siblings. *American Journal of Orthopsychiatry*, 35, 747–753.
- Large, M., Sharma, S., Compton, M. T., Slade, T., & Nielssen, O. (2011). Cannabis use and earlier onset of psychosis: A systematic meta-analysis. *Archives of General Psychiatry*, doi: 10.1001/archgenpsychiatry.2011.5
- Leucht, S., Arbter, D., Engel, R. R., Kissling, W., & Davis, J. M. (2008). How effective are second-generation antipsychotic drugs? A meta-analysis of placebo-controlled trials. *Mol Psychiatry*, 14(4), 429–447.
- Leucht, S., Cipriani, A., Spineli, L., Mavridis, D., Örey, D., Richter, F., ... Davis, J. M. (2013). Comparative efficacy and tolerability of 15 antipsychotic drugs in schizophrenia: a multiple-treatments meta-analysis. *The Lancet*, 382(9896), 951–962. doi: 10.1016/S0140-6736(13)60733-3
- Leucht, S., Corves, C., Arbter, D., Engel, R. R., Li, C., & Davis, J. M. (2009). Second-generation versus first-generation antipsychotic drugs for schizophrenia: a meta-analysis. *Lancet*, 373, 31–41.
- Leucht, S., Tardy, M., Komossa, K., Heres, S., Kissling, W., & Davis, J. M. (2009). Maintenance treatment with antipsychotic drugs for schizophrenia. (1469-493X (Electronic)).
- Liberman, R. P., Eckman, T. A., Kopelowicz, A., & Stolar, D. (2000). *Friendship and intimacy module*. Camarillo, CA: Psychiatric Rehabilitation Consultants.
- Liberman, R. P., Wallace, C. J., Blackwell, G., Kopelowicz, J. V., et al. (1998). Skills training versus psychosocial occupational therapy for persons with persistent schizophrenia. *American Journal of Psychiatry*, 155, 1087–1091.
- Lieberman, J. A., Stroup, T. S., McEvoy, J. P., Swartz, M. S., Rosenheck, R. A., Perkins, D. O., ... the Clinical Antipsychotic Trials of Intervention Effectiveness, I. (2005). Effectiveness of antipsychotic drugs in patients with chronic schizophrenia. *The New England Journal of Medicine*, 353(12), 1209–1223.
- Lim, K. O., Adalsteinsson, E., Spielman, D., Sullivan, E. V., Rosenbloom, M. J., & Pfefferman, A. (1998). Proton magnetic resonance spectroscopic imaging of cortical gray and white matter in schizophrenia. *Archives of General Psychiatry*, 55, 346–353.
- Lin, A., Wood, S. J., Nelson, B., Beavan, A., McGorry, P., & Yung, A. R. (2015). Outcomes of nontransitioned cases in a sample at ultra-high risk for psychosis. *American Journal of Psychiatry*, 172(3), 249–258. doi:10.1176/appi.ajp.2014.13030418
- Lopez, S. R., Nelson, K. A., Snyder, K. S., & Mintz, J. (1999). Attributions and affective reactions of family members and course of schizophrenia. *Journal of Abnormal Psychology*, 108, 307–314.
- MacDonald, A. W., III, & Carter, C. S. (2003). Event-related fMRI study of context processing in dorsolateral prefrontal cortex of patients with schizophrenia. *Journal of Abnormal Psychology*, 112, 689–697.
- MacDonald, A. W., III, & Chafee, D. (2006). Translational and developmental perspective on N-methyl-D-aspartate synaptic deficits in schizophrenia. *Development and Psychopathology*, 18, 853–876.
- Malaspina, D., Goetz, R. R., Yale, S., et al. (2000). Relation of familial schizophrenia to negative symptoms but not to the deficit syndrome. *American Journal of Psychiatry*, 157, 994–1003.
- Marder, S. R., Wirshing, W. C., Glynn, S. M., Wirshing, D. A., Mintz, J., & Liberman, R. P. (1999). Risperidone and haloperidol in maintenance treatment: Interactions with psychosocial treatments. *Schizophrenia Research*, 36, 288.

- Marsh, P. J., Luckett, G., Russell, T., Coltheart, M., & Green, M. J. (2012). Effects of facial emotion recognition remediation on visual scanning of novel face stimuli. *Schizophrenia Research*, 141(2–3), 234–240. doi: 10.1016/j.schres.2012.08.006
- Marshall, M., Lewis, S., Lockwood, A., Drake, R., Jones, P., & Croudace, T. (2005). Association between duration of untreated psychosis and outcome in cohorts of first-episode patients: A systematic review. *Archives of General Psychiatry*, 62(9), 975–983. doi: 10.1001/archpsyc.62.9.975
- Mathew, I., Gardin, T. M., Tandon, N., Eack, S., Francis, A. N., Seidman, L. J., et al. (2014). Medial temporal lobe structures and hippocampal subfields in psychotic disorders: Findings from the Bipolar-Schizophrenia Network on Intermediate Phenotypes (B-SNIP) study. *Journal of the American Medical Association, Psychiatry*, 71, 769–777.
- McEvoy, J. P., Johnson, J., Perkins, D., Lieberman, J. A., Hamer, R. M., Keefe, R. S. E., et al. (2006). Insight in first episode psychosis. *Psychological Medicine*, 36, 1385–1393.
- McEvoy, J. P., Lieberman, J. A., Stroup, T. S., Davis, S. M., Meltzer, H. Y., Rosenheck, R. A., . . . for the CATIE Investigators. (2006). Effectiveness of clozapine versus olanzapine, quetiapine and risperidone in patients with chronic schizophrenia who did not respond to prior atypical antipsychotic treatment. *American Journal of Psychiatry*, 163, 600–610.
- McFarlane, W. R., Lukens, E., Link, B., Dushay, R., Deakins, S., Newmark, M., Dunne, E. J., Horen, B., & Toran, J. (1995). Multiple-family groups and psychoeducation in the treatment of schizophrenia. *Archives of General Psychiatry*, 52, 679–687.
- McGhie, A., & Chapman, I. S. (1961). Disorders of attention and perception in early schizophrenia. *British Journal of Medical Psychology*, 34, 103–116.
- McGlashan, T. H., & Hoffman, R. E. (2000). Schizophrenia as a disorder of developmentally reduced synaptic connectivity. *Archives of General Psychiatry*, 57, 637–648.
- McGrath, J. J., Petersen, L., Agerbo, E., Mors, O., Mortensen, P., & Pedersen, C. (2014). A comprehensive assessment of parental age and psychiatric disorders. *JAMA Psychiatry*, 71(3), 301–309. doi: 10.1001/jamapsychiatry.2013.4081
- McGurk, S. R., Mueser, K. T., Feldman, K., Wolfe, R., & Pascaris, A. (2007). Cognitive training for supported employment: 2–3 year outcomes of a randomized controlled trial. *American Journal of Psychiatry*, 164(3), 377–379.
- McGurk, S. R., Mueser, K. T., Xie, H., Welsh, J., Kaiser, S., Drake, R. E., . . . McHugo, G. J. (2015). Cognitive enhancement treatment for people with mental illness who do not respond to supported employment: A randomized controlled trial. *American Journal of Psychiatry*, 172(9), 852–861. doi: 10.1176/appi.ajp.2015.14030374
- McGurk, S. R., Twamley, E. W., Sitzer, D. I., McHugo, G. J., & Mueser, K. T. (2007). A meta-analysis of cognitive remediation in schizophrenia. *American Journal of Psychiatry*, 164, 1791–1802.
- McNeil, T. F., Cantor-Graae, E., & Weinberger, D. R. (2000). Relationship of obstetric complications and differences in size of brain structures in monozygotic twin pairs discordant for schizophrenia. *American Journal of Psychiatry*, 157, 203–212.
- Mednick, S. A., Cudeck, R., Griffith, J. J., Talovic, S. A., & Schlusinger, F. (1984). The Danish high-risk project: recent methods and findings. In N. F. Watt (Ed.), *Children at risk for schizophrenia. A longitudinal perspective* (pp. 21–42). Cambridge: Cambridge University Press.
- Mednick, S. A., Huttonen, M. O., & Machon, R. A. (1994). Prenatal influenza infections and adult schizophrenia. *Schizophrenia Bulletin*, 20, 263–268.
- Mednick, S. A., Machon, R., Huttonen, M. O., & Bonett, D. (1988). Fetal viral infection and adult schizophrenia. *Archives of General Psychiatry*, 45, 189–192.
- Meier, M. H., Caspi, A., Reichenberg, A., Keefe, R. S. E., Fisher, H. L., Harrington, H., . . . Moffitt, T. E. (2014). Neuropsychological decline in schizophrenia from the premorbid to the postonset period: Evidence from a population-representative longitudinal study. *American Journal of Psychiatry*, 171(1), 91–101. doi: 10.1176/appi.ajp.2013.12111438
- Meijer, C. J., Koeter, M. W. J., Sprangers, M. A. G., & Schene, A. H. (2009). Predictors of general quality of life and the mediating role of health related quality of life in patients with schizophrenia. *Social Psychiatry and Psychiatric Epidemiology*, 44, 361–368.
- Menezes, N. M., Arenovich, T., & Zipursky, R. B. (2006). A systematic review of longitudinal outcome studies of first-episode psychosis. *Psychological Medicine*, 36, 1349–1362.
- Mental Health Commission. (2012). *Blueprint ii: How things need to be*. Wellington, New Zealand.
- Mesholam-Gately, R., Giuliano, A. J., Goff, K. P., Faraone, S. V., & Seidman, L. J. (2009). Neurocognition in first-episode schizophrenia: A meta-analytic review. *Neuropsychology*, 23(3), 315–336.
- Messinger, J. W., Treméau, F., Antonius, D., Mendelsohn, E., Prudent, V., Stanfore, A. D., & Malaspina, D. (2011). Avolition and expressive deficits capture negative symptom phenomenology: Implications for the DSM-5 and schizophrenia research. *Clinical Psychology Review*, 31, 161–168.
- Milev, P., Ho, B. C., Arndt, S., & Andreasen, N. C. (2005). Predictive values of neurocognition and negative symptoms on functional outcome in schizophrenia: A longitudinal first-episode study with 7-year follow-up. *American Journal of Psychiatry*, 162(3), 495–506.
- Mirsky, A. F., Bieliauskas, L. A., Duncan, C. C., & French, L. M. (2013). Letter to the editor. *Schizophrenia Research*, 148(1–3), 186–187.
- Morgan, V. A., Waterreus, A., Jablensky, A., Mackinnon, A., McGrath, J. J., Carr, V., . . . Saw, S. (2011). *People living with psychotic illness 2010*. Canberra ACT 2601: Commonwealth of Australia, Retrieved from [www.health.gov.au/internet/main/publishing.nsf/Content/mental-pubs-p-psych10](http://www.health.gov.au/internet/main/publishing.nsf/Content/mental-pubs-p-psych10).
- Morrison, A. P., French, P., Stewart, S. L. K., Birchwood, M., Fowler, D., Gumley, A. I., . . . Dunn, G. (2012). Early detection and intervention evaluation for people at risk of psychosis: multisite randomised controlled trial. *BMJ*, 344, e2233. doi: 10.1136/bmj.e2233

- Morrison, A. P., French, P., Walford, L., Lewis, S. W., Kilcommins, A., Green, J., ... Bentall, R. P. (2004). Cognitive therapy for the prevention of psychosis in people at ultra-high risk. *British Journal of Psychiatry*, 185, 291–297.
- Morrison, A. P., Turkington, D., Pyle, M., Spencer, H., Brabban, A., Dunn, G., ... Hutton, P. (2014). Cognitive therapy for people with schizophrenia spectrum disorders not taking antipsychotic drugs: a single-blind randomised controlled trial. *Lancet*. doi: doi.org/10.1016/S0140-6736(13)62246-1
- Mueser, K. T., Bond, G. R., Drake, R. E., & Resnick, S. G. (1998). Models of community care for severe mental illness: A review of research on case management. *Schizophrenia Bulletin*, 24, 37–74.
- Mullin, K., Gupta, P., Compton, M. T., Nielssen, O., Harris, A., & Large, M. (2012). Does giving up substance use work for patients with psychosis? A systematic meta-analysis. *Australian and New Zealand Journal of Psychiatry*, 00. doi: 10.1177/0004867412440192
- Myin-Germeys, I., van Os, J., Schwartz, J. E., et al. (2001). Emotional reactivity to daily life stress in schizophrenia. *Archives of General Psychiatry*, 58, 1137–1144.
- National Collaborating Centre for Mental Health. (2014). *Psychosis and schizophrenia in adults: Treatment and management*. National Institute for Health and Care Excellence.
- Neale, J. M., & Oltmanns, T. (1980). *Schizophrenia*. New York: John Wiley & Sons.
- Nelson, B., Yuen, H. P., Wood, S. J., Lin, A., Spiliotacopoulos, D., Bruxner, S. M., ... Yung, A. R. (2013). Long-term follow-up of a group at ultra high risk ('Prodromal') for psychosis. The PACE 400 Study. *JAMA Psychiatry*. doi:10.1001/jamapsychiatry.2013.1270
- Nelson, M. D., Saykin, A. J., Flashman, L. A., & Riordin, H. J. (1998). Hippocampal volume reduction in schizophrenia as assessed by magnetic resonance imaging: A meta analytic study. *Archives of General Psychiatry*, 55, 433–440.
- O'Donnell, P., & Grace, A. A. (1998). Dysfunctions in multiple interrelated systems as the neurobiological bases of schizophrenic symptom clusters. *Schizophrenia Bulletin*, 24, 267–283.
- O'Neal, J. M. (1984). First person account: Finding myself and loving it. *Schizophrenia Bulletin*, 10, 109–110.
- Ohtani, T., Levitt, J. J., Nestor, P. G., Kawashima, T., Asami, T., Shenton, M. E., et al. (2014). Prefrontal cortex volume deficit in schizophrenia: A new look using 3T MRI with manual parcellation. *Schizophrenia Research*, 152(1), 184–190.
- Olabi, B., Ellison-Wright, I., McIntosh, A. M., Wood, S. J., Bullmore, E., & Lawrie, S. M. (2011). Are there progressive brain changes in schizophrenia? A meta-analysis of structural magnetic resonance imaging studies. *Biological Psychiatry*, 70, 88–96.
- Palmer, B. A., Pankratz, V. S., & Bostwick, J. M. (2005). The lifetime risk of suicide in schizophrenia: A reexamination. *Archives of General Psychiatry*, 62(3), 247–253.
- Pantelis, C., Velakoulis, D., McGorry, P. D., et al. (2003). Neuroanatomical abnormalities before and after onset of psychosis: A cross-sectional and longitudinal MRI comparison. *The Lancet*, 361, 281–288.
- Pathania, M., Davenport, E. C., Muir, J., Sheehan, D. F., Lopez-Domenech, G., & Kittler, J. T. (2014). The autism and schizophrenia associated gene CYFIP1 is critical for the maintenance of dendritic complexity and the stabilization of mature spines. *Translational Psychiatry*, 4, e374.
- Pathmanandavel, K., Starling, J., Merheb, V., Ramanathan, S., Sinmaz, N., Dale, R. C., & Brilot, F. (2015). Antibodies to surface dopamine-2 receptor and N-methyl-D-aspartate receptor in the first episode of acute psychosis in children. *Biological Psychiatry*, 77(6), 537–547. doi: http://dx.doi.org/10.1016/j.biopsych.2014.07.014
- Penn, D. L., & Mueser, K. T. (1996). Research update on the psychosocial treatment of schizophrenia. *American Journal of Psychiatry*, 153, 607–617.
- Penzes, P., Cahill, M. E., Jones, K. A., VanLeeuwen, J. E., & Woolfrey, K. M. (2011). Dendritic spine pathology in neuropsychiatric disorders. *Nature Neuroscience*, 14, 285–293.
- Pettersson-Yeo, W., Allen, P., Benetti, S., McGuire, P., & Mechelli, A. (2011). Dysconnectivity in schizophrenia: where are we now? *Neuroscience Biobehavioral Reviews*, 35(5), 1110–1124.
- Phillips, L. J., Francey, S. M., Edwards, J., & McMurray, N. (2007). Stress and psychosis: towards the development of new models of investigation. *Clinical Psychology Review*, 27, 307–317.
- Pilling, S., Bebbington, P., Kuipers, E., Garety, P., Geddes, L., Martindale, B., Orbach, G., & Morgan, C. (2002). Psychological treatments in schizophrenia: II. Meta-analyses of randomized controlled trials of social skills training and cognitive remediation. *Psychological Medicine*, 32, 783–791.
- Potkin, S. G., Alva, G., Fleming, K., et al. (2002). A PET study of the pathophysiology of negative symptoms in schizophrenia. *American Journal of Psychiatry*, 159, 227–237.
- Rapoport, J. L., Giedd, J., & Gogtay, N. (2012). Neurodevelopmental model of schizophrenia: Update 2012. *Molecular Psychiatry*, 17, 1228–1238.
- Rasic, D., Hajek, T., Alda, M., & Uher, R. (2014). Risk of mental illness in offspring of parents with schizophrenia, bipolar disorder, and major depressive disorder: A meta-analysis of family high-risk studies. *Schizophrenia Bulletin*, 40(1), 28–38.
- Rees, E., O'Donovan, M. C., & Owen, M. J. (2015). Genetics of schizophrenia. *Current Opinion in Behavioural Sciences*, 2, 8–14. doi: http://dx.doi.org/10.1016/j.cobeha.2014.07.001
- Regland, B., Johansson, B.V., Grenfeldt, B., Hjelmgren, L.T., & Medhus, M. (1995). Homocysteinemia is a common feature of schizophrenia. *Journal of Neural Transmission: General Section*, 100, 165–169.
- Reichenberg, A., Caspi, A., Harrington, H., Houts, R., Keefe, R. S. E., Murray, R. M., ... Moffit, T. E. (2010). Static and dynamic cognitive deficits in childhood preceding adult schizophrenia: A 30-year study. *American Journal of Psychiatry*, 167(2), 160–169. doi: 10.1176/appi.ajp.2009.09040574

- Restifo, K., Harkavy-Friedman, J. M., & Shrout, P. E. (2009). Suicidal behaviour in schizophrenia. A test of the demoralization hypothesis. *Journal of Nervous & Mental Disease*, 197, 147–153.
- Rieder, R. O., Mann, L. S., Weinberger, D. R., van Kammen, D. P., & Post, R. M. (1983). Computer tomographic scans in patients with schizophrenia, schizoaffective, and bipolar affective disorder. *Archives of General Psychiatry*, 40, 735–739.
- Roberts, D. L., & Penn, D. L. (2009). Social cognition and interaction training (SCIT) for outpatients with schizophrenia: A preliminary study. *Psychiatry Research*, 166, 141–147.
- Rosenfarb, I. S., Goldstein, M. J., Mintz, J., & Neuchterlein, K. H. (1994). Expressed emotion and subclinical psychopathology observable within transactions between schizophrenics and their family members. *Journal of Abnormal Psychology*, 104, 259–267.
- Saha, S., Chant, D., & McGrath, J. (2007). A systematic review of mortality in schizophrenia: Is the differential mortality gap worsening over time? *Archives of General Psychiatry*, 64, 1123–1131.
- Saha, S., Chant, D., Welham, J., & McGrath, J. (2005). A systematic review of the prevalence of schizophrenia. *PLoS Medicine*, 2(5), e141.
- Saks, E. R. (2007). *The center cannot hold: My journey through madness*. New York: Hyperion.
- Salem, J. E., & Kring, A. M. (1998). The role of gender differences in the reduction of etiologic heterogeneity in schizophrenia. *Clinical Psychology Review*, 18, 795–819.
- Sartorius, N., Shapiro, R., & Jablonsky, A. (1974). The international pilot study of schizophrenia. *Schizophrenia Bulletin*, 2, 21–35.
- Schizophrenia Working Group of the Psychiatric Genomics Consortium. (2014). Biological insights from 108 schizophrenia-associated genetic loci. *Nature*, 511, 421–427. doi:10.1038/nature13595
- Schmidt, S. J., Mueller, D. R., & Roder, V. (2011). Social cognition as a mediator variable between neurocognition and functional outcome in schizophrenia: Empirical review and new results by structural equation modeling. *Schizophrenia Bulletin*, 37 (suppl 2), S41–S54. doi: 10.1093/schbul/sbr079
- Sekar, A., Bialas, A. R., de Rivera, H., Davis, A., Hammond, T. R., Kamitaki, N., . . . McCarroll, S. A. (2016). Schizophrenia risk from complex variation of complement component 4. *Nature*, advance online publication. doi: 10.1038/nature16549
- Sensky, T., Turkington, D., Kingdon, D., et al. (2000). A randomized controlled trial of cognitive-behavioural therapy for persistent symptoms in schizophrenia resistant to medication. *Archives of General Psychiatry*, 57, 165–172.
- Sevy, S., Nathanson, K., Visweswarajah, H., & Amador, X. (2004). The relationship between insight and symptoms in schizophrenia. *Comprehensive Psychiatry*, 45(1), 16–19. doi:10.1016/j.comppsy.2003.09.002
- Siegel, S. J., Irani, F., Brensinger, C. M., Kohler, C. G., Bilker, W. B., Ragland, J. D., et al. (2006). Prognostic variables at intake and long-term level of function in schizophrenia. *American Journal of Psychiatry*, 163(3), 433–441.
- Simpson, E. H., Kellendonk, C., & Kandel, E. (2010). A possible role for the striatum in the pathogenesis of the cognitive symptoms of schizophrenia. *Neuron*, 65, 585–596. doi: DOI 10.1016/j.neuron.2010.02.014
- Singh, S. P., Harley, K., & Suhail, K. (2013). Cultural specificity of emotional overinvolvement: A systematic review. *Schizophrenia Bulletin*, 39(2), 449–463.
- Slade, M. (2009). *Personal recovery and mental illness: a guide for mental health professionals*. Cambridge: Cambridge University Press.
- Slifstein, M., van de Giessen, E., Van Snellenberg, J., et al. (2015). Deficits in prefrontal cortical and extrastriatal dopamine release in schizophrenia: A positron emission tomographic functional magnetic resonance imaging study. *JAMA Psychiatry*, 72(4), 316–324. doi: 10.1001/jamapsychiatry.2014.2414
- St Clair, D., Xu, M., Wang, P., Yu, Y., Fang, Y., Zhang, F., . . . He, L. (2005). Rates of adult schizophrenia following prenatal exposure to the Chinese famine of 1959–61. *JAMA*, 294(557–562).
- Steen, R. G., Mull, C., McClure, R., Hamer, R. M., & Lieberman, J. A. (2006). Brain volume in first-episode schizophrenia: Systematic review and meta-analysis of magnetic resonance imaging studies. *British Journal of Psychiatry*, 188, 510–518.
- Stein, L. I., & Test, M. A. (1980). Alternative to mental hospital treatment: I. Conceptual model, treatment program, and clinical evaluation. *Archives of General Psychiatry*, 37, 392–397.
- Steiner, J., Walter, M., Glanz, W., Sarnyai, Z., Bernstein, H. G., Vielhaber, S., et al. (2013). Increased prevalence of diverse N-methyl-D-aspartate glutamate receptor antibodies in patients with an initial diagnosis of schizophrenia: Specific relevance of IgG NR1a antibodies for distinction from N-methyl-D-aspartate glutamate receptor encephalitis. *Journal of the American Medical Association Psychiatry*, 70(3), 271–278.
- Suddath, R. L., Christison, G. W., Torrey, E. F., Cassanova, M. F., Weinberger, D. R., et al. (1990). Anatomical abnormalities in the brains of monozygotic twins discordant for schizophrenia. *New England Journal of Medicine*, 322, 789–793.
- Sullivan, P. F. (2005). The genetics of schizophrenia. *PLoS Medicine*, 2(7), 614–618.
- Sullivan, P. F., Kendler, K. S., & Neale, M. C. (2003). Schizophrenia as a complex trait: evidence from a meta-analysis of twin studies. *Archives of General Psychiatry*, 60, 1187–1192.
- Sun, D., Phillips, L., Velakoulis, D., Yung, A., McGorry, P. D., Wood, S. J., . . . Pantelis, C. (2009). Progressive brain structural changes mapped as psychosis develops in 'at risk' individuals. *Schizophrenia Research*, 108(1–3), 85–92. doi: http://dx.doi.org/10.1016/j.schres.2008.11.026
- Surles, R. C., Blanch, A. K., Shern, D. L., & Donahue, S. A. (1992). Case management as a strategy for systems change. *Health Affairs*, 11, 151–163.
- Susser, E., Neugebauer, R., Hoek, H. W., Brown, A. S., Lin, S., Labovitz, D., & Gorman, J. M. (1996). Schizophrenia After Prenatal Famine: Further Evidence. *Archives of General Psychiatry*, 53(1), 25–31. doi: 10.1001/archpsyc.1996.01830010027005

- Sweet, R. A., Mulsant, B. H., Gupta, B., Rifai, A. H., Pasternak, R. E., et al. (1995). Duration of neuroleptic treatment and prevalence of tardive dyskinesia in late life. *Archives of General Psychiatry*, 52, 478–486.
- Tsai, G., Parssani, L. A., Slusher, B. S., Carter, R., Baer, L., et al. (1995). Abnormal excitatory neurotransmitter metabolism in schizophrenic brains. *Archives of General Psychiatry*, 52, 829–836.
- Turkington, D., Kingdom, D., & Turner, T. (2002). Effectiveness of a brief cognitive-behavioural intervention in the treatment of schizophrenia. *British Journal of Psychiatry*, 180, 523–527.
- Unschuld, P. G., Buchholz, A. S., Varvaris, M., van Zijl, P. C., Ross, C. A., Pekar, J. J., et al. (2014). Prefrontal brain network connectivity indicates degree of both schizophrenia risk and cognitive dysfunction. *Schizophrenia Bulletin*, 40(3), 653–664.
- Valencia, M., Racon, M. L., Juarez, F., & Murow, E. (2007). A psychosocial skills training approach in Mexican outpatients with schizophrenia. *Psychological Medicine*, 37, 1393–1402.
- van der Gaag, M., Nieman, D. H., Rietdijk, J., Dragt, S., Ising, H. K., Klaassen, R. M. C., . . . Linszen, D. H. (2012). Cognitive behavioural therapy for subjects at ultrahigh risk for developing psychosis: A randomized controlled clinical trial. *Schizophrenia Bulletin*. doi: 10.1093/schbul/sbs105
- van Erp, T. G. M., Saleh, P. A., Huttunen, M., et al. (2004). Hippocampal volumes in schizophrenic twins. *Archives of General Psychiatry*, 61, 346–353.
- van Os, J., Kenis, G., & Rutten, B. P. (2010). The environment and schizophrenia. *Nature*, 468, 203–212.
- Velakoulis, D., Pantelis, C., McGorry, P. D., Dudgeon, P., Brewer, W., et al. (1999). Hippocampal volume in first episode psychoses and chronic schizophrenia: A high resolution magnetic resonance imaging study. *Archives of General Psychiatry*, 56, 133–141.
- Ventura, J., Neuchterlein, K. H., Lukoff, D., & Hardesty, J. D. (1989). A prospective study of stressful life events and schizophrenic relapse. *Journal of Abnormal Psychology*, 98, 407–411.
- Vitaliani, R., Mason, W., Ances, B., Zwerdling, T., Jiang, Z., & Dalmau, J. (2005). Paraneoplastic encephalitis, psychiatric symptoms, and hypoventilation in ovarian teratoma. *Annals of Neurology*, 58(4), 594–604.
- Volk, D. W., Austin, M. C., Pierri, J. N., et al. (2000). Decreased glutamic acid decarboxylase67 messenger RNA expression in a subset of prefrontal cortical gamma-aminobutyric acid neurons in subjects with schizophrenia. *Archives of General Psychiatry*, 57, 237–248.
- Waghorn, G., Saha, S., Harvey, C., Morgan, V. A., Waterreus, A., Bush, R., . . . McGrath, J. J. (2012). 'Earning and learning' in those with psychotic disorders: The second Australian national survey of psychosis. *Australian and New Zealand Journal of Psychiatry*, 46(8), 774–785. doi: 10.1177/0004867412452015
- Walker, E. F., Davis, D. M., & Savoie, T. D. (1994). Neuromotor precursors of schizophrenia. *Schizophrenia Bulletin*, 20, 441–451.
- Walker, E. F., Grimes, K. E., Davis, D. M., & Adina, J. (1993). Childhood precursors of schizophrenia: Facial expressions of emotion. *American Journal of Psychiatry*, 150, 1654–1660.
- Walker, E. F., Kestler, L., Bollini, A., & Hochman, K. (2004). Schizophrenia: Etiology and course. *Annual Review of Psychology*, 55, 401–430.
- Walker, E. F., Mittal, V., & Tessner, K. (2008). Stress and the hypothalamic pituitary adrenal axis in the developmental course of schizophrenia. *Annual Review of Clinical Psychology*, 4, 189–216.
- Walker, E. F., Trotman, H. D., Pearce, B. D., Addington, J., Cadenhead, K. S., Cornblatt, B. A., et al. (2013). Cortisol levels and risk for psychosis: initial findings from the North American prodrome longitudinal study. *Biological Psychiatry*, 74(6), 410–417.
- Watt, N. F. (1974). Childhood and adolescent roots of schizophrenia. In D. Ricks, A. Thomas & M. Roll (Eds.), *Life history research in psychopathology* (Vol. 3). Minneapolis: University of Minnesota Press.
- Watt, N. F., Stolorow, R. D., Lubensky, A. W., & McClelland, D. C. (1970). School adjustment and behavior of children hospitalized for schizophrenia as adults. *American Journal of Orthopsychiatry*, 40, 637–657.
- Weder, N. D., Muralee, S., Penland, H., & Tampi, R. R. (2008). Catatonia: A review. *Annals of Clinical Psychiatry*, 20(2), 97–107.
- Wegener, S., Redoblado-Hodge, A., Lucas, S., Fitzgerald, D., Harris, A., & Brennan, J. (2005). Relative contributions of psychiatric symptoms and neuropsychological functioning to quality of life in first episode psychosis. *Australian and New Zealand Journal of Psychiatry*, 38(6), 487–492.
- Weinberger, D. R. (1987). Implications of normal brain development for the pathogenesis of schizophrenia. *Archives of General Psychiatry*, 44, 660–669.
- Weinberger, D. R., Berman, K. F., & Illowsky, B. P. (1988). Physiological dysfunction of dorsolateral prefrontal cortex in schizophrenia: 3. A new cohort and evidence for a monoaminergic mechanism. *Archives of General Psychiatry*, 45, 609–615.
- Weinberger, D. R., Cannon-Spoor, H. E., Potkin, S. G., & Wyatt, R. J. (1980). Poor premorbid adjustment and CT scan abnormalities in chronic schizophrenia. *American Journal of Psychiatry*, 137, 1410–1413.
- Weisman, A. G., Neuchterlein, K. H., Goldstein, M. J., & Snyder, K. S. (1998). Expressed emotion, attributions, and schizophrenia symptom dimensions. *Journal of Abnormal Psychology*, 107, 355–359.
- Whitford, T. J., Ford, J. M., Mathalon, D. H., Kubicki, M., & Shenton, M. E. (2012). Schizophrenia, myelination, and delayed corollary discharges: A hypothesis. *Schizophrenia Bulletin*, 38(3), 486–494. doi: 10.1093/schbul/sbq105
- Woodberry, K. A., Giuliano, A. J., & Seidman, L. J. (2008). Premorbid IQ in schizophrenia: A meta-analytic review. *American Journal of Psychiatry*, 165, 579–587.
- World Health Organization. (2013). *Mental health action plan 2013-2020*. Geneva: World Health Organization.
- Wright, I. A., Rabe-Hesketh, S., Woodruff, P. W., Davis, A. S., Murray, R. M., & Bullmore, E. T. (2000). Meta-analysis of regional brain volumes in schizophrenia. *American Journal of Psychiatry*, 157, 16–25.

- Wunderink, L., Nieboer, R. M., Wiersma, D., Sytema, S., & Nienhuis, F. J. (2013). Recovery in remitted first-episode psychosis at 7 years of follow-up of an early dose reduction/discontinuation or maintenance treatment strategy: Long-term follow-up of a 2-year randomized clinical trial. *JAMA Psychiatry*, 70(9), 913–920. doi: 10.1001/jamapsychiatry.2013.19
- Wykes, T., Huddy, V., Cellard, C., McGurk, S., & Czobor, P. (2011). A meta-analysis of cognitive remediation for schizophrenia: Methodology and effect sizes. *American Journal of Psychiatry*, 168, 472–485.
- Wykes, T., Steel, C., Everitt, T., & Tarrier, N. (2008). Cognitive behavior therapy for schizophrenia: Effect sizes, clinical models, and methodological rigor. *Schizophrenia Bulletin*, 34, 523–537.
- Xia, J., Merinder, L. B., & Belgamwar, M. R. (2011). Psychoeducation for schizophrenia. *Schizophrenia Bulletin*, 37, 21–22.
- Yung, A. R., & McGorry, P. D. (1996). The initial prodrome in psychosis: descriptive and qualitative aspects. *Australian and New Zealand Journal of Psychiatry*, 30, 587–599.
- Yung, A. R., McGorry, P. D., McFarlane, C. A., & Patton, G. (1995). The PACE Clinic: Development of a clinical service for young people at high risk of psychosis. *Australian Psychiatry*, 3, 345–349.
- Yung, A. R., Woods, S. W., Ruhrmann, S., Addington, J., Schultze-Lutter, F., Cornblatt, B. A., et al. (2012). Whither the attenuated psychosis syndrome? *Schizophrenia Bulletin*, 38(6), 1130–1134.
- Yung, A. R., Yuen, H. P., McGorry, P. D., Phillips, L., Kelly, D., Dell’Olio, M., . . . Buckby, J. (2005). Mapping the onset of psychosis: the Comprehensive Assessment of At-Risk Mental States. *Australian and New Zealand Journal of Psychiatry*, 39(11–12), 964–971.

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## CHAPTER 7

# Substance use disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 7.1** describe the epidemiology and symptoms associated with substance use disorders
  - 7.2** understand the major aetiological factors for substance use disorders, including genetic factors, neurobiological factors, mood and expectancy effects and sociocultural factors
  - 7.3** describe the approaches to treating substance use disorders, including psychological treatments, medications and drug substitution treatments
  - 7.4** delineate the major approaches to preventing substance use disorders.
-

## OPENING SCENARIO

Susan is a young woman who goes out on most weekends with her friends. Susan usually drinks alcohol when she goes out. She doesn't usually drink much during the rest of the week (2–3 drinks a day) but on weekend nights Susan regularly drinks around 20 drinks over a period of 8–10 hours. Sometimes she drinks so much on the weekend that she is unable to get home. Susan's friends have noticed that she is drinking more and she is drinking a lot more than they do. Susan's friends and family are concerned for her as there have been a number of nights when she couldn't remember what she did and how she got home. She has messages on her phone from people she does not remember meeting. She was recently hospitalised after falling and breaking her wrist while trying to walk home . . . She remembers starting the night at her local bar listening to music and thinking that she would only have three drinks. At the end of the third drink she didn't stop drinking and by the end of the night she could not manage the stairs, tripped and fell. When she spoke to the medical staff at the hospital she realised that she had been drinking a lot more than she used to. The doctor told her to cut down her drinking. She didn't ask for help for her drinking as she didn't think she had a problem, but that she had simply slipped and broken her wrist. She has now started to notice that she has increasingly strong urges to drink during the week and has been drinking about four to five drinks every evening. She still feels her life and drinking are okay, no different from her friends and that she can stop whenever she wants.

She feels safe as she is only drinking, whereas her friend Jane is still at university and takes ecstasy most weekends when she goes out with friends. Despite trying to limit the amount she spends on drugs, she often spends money meant for food on drugs for a night out. Jane is also skipping classes on Mondays to recover from her weekends of drug use and hasn't finished her degree yet.

### QUESTIONS

1. Why do some people develop problems with alcohol and drugs while others do not?
2. How should we best respond and treat problems associated with alcohol and drug use?

## Introduction

People have used various substances in the hope of reducing pain, changing mood or altering states of consciousness for centuries. Around the world, almost all people use one or more substances that affect the central nervous system, relieving physical and mental anguish or producing euphoria. Not everyone develops problems with alcohol and drugs, but some will. Why do some people develop problems with alcohol and drugs while others do not? How do we best prevent and treat alcohol and drug use disorders? The answers to these important questions are determined by multiple interacting factors, the unravelling of which provides a challenge to **addiction** research and addiction treatment. Substance use disorders are one way of considering some of the harms and problems caused by substances and they are the subject of this chapter. Before we go on, it's worth remembering that the majority of harms from substances are not caused by substance use disorders.

## 7.1 Clinical descriptions, prevalence and effects of substance use disorders

**LEARNING OUTCOME 7.1** Describe the epidemiology and symptoms associated with substance use disorders.

Australians use drugs to wake up (coffee or tea), to stay alert throughout the day (cigarettes, soft drinks), to relax (alcohol) and to reduce pain (paracetamol). The widespread availability and frequent use of drugs set the stage for the potential abuse of drugs, the topic of this chapter.

## DSM-5

### DSM-5 criteria for substance use disorder

People with substance use disorder undertake a problematic pattern of use that impairs functioning. The individual experiences two or more of the following symptoms within a one-year period:

- failure to meet obligations
- repeated use in situations where it is physically dangerous
- repeated relationship problems
- continued use despite problems caused by the substance
- tolerance
- withdrawal
- substance taken for a longer time or in greater amounts than intended
- efforts to reduce or control use do not work
- much time spent obtaining, using or recovering from the substance
- social, hobbies or work activities given up or reduced
- craving to use the substance is strong.

Recent data on the frequency of drug use are presented in table 7.1. These figures do not represent the frequency of substance use disorders but simply indicate the pervasiveness of drug and alcohol use in Australia. Alcohol remains the most commonly used substance, with nearly 80 percent of Australians over the age of 14 reporting alcohol use of some kind in the previous 12 months (Australian Institute of Health and Welfare, AIHW, 2014). In 2013, 15.6 percent of people aged 14 or older had consumed 11 or more standard drinks on a single drinking occasion in the past 12 months. In Australia, 12.8 percent of the population are current daily smokers and nearly a quarter of the population (24 percent) are ex-smokers. In 2013, nearly 2.9 million people (15 percent) over the age of 14 in Australia reported having used an illicit drug in the past 12 months. Cannabis was the most frequently used, with 10.2 percent of the Australian population over the age of 14 reporting cannabis use in the past 12 months.

**TABLE 7.1** Percentage of Australian population reporting drug use in past 12 months (2013)

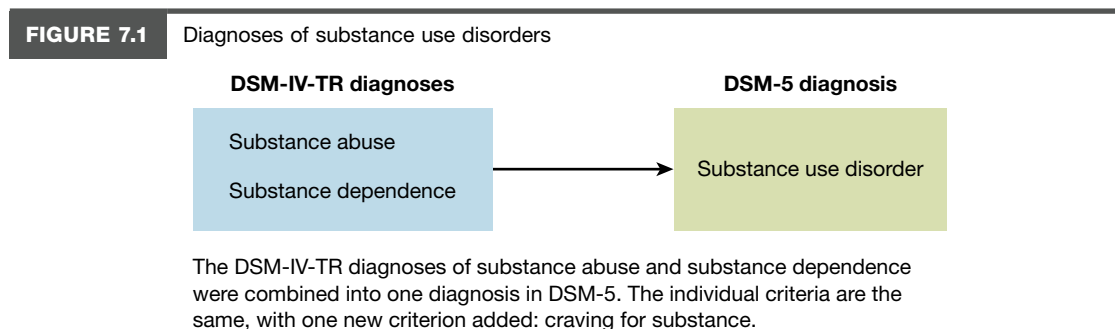
Substance	Percentage reporting use
Alcohol	78.2
Cannabis	10.2
Ecstasy	2.5
Methamphetamines	2.1
Cocaine	2.1
Heroin	0.1
Hallucinogens	1.3
Inhalants	0.8
Non-medical use of pharmaceuticals	4.7

**Note:** Data are percentages of people in Australia aged 14 years and over. Non-medical use of pharmaceuticals refers to the use of pain medicines (3.3 percent), tranquilisers (1.6 percent), steroids (0.1 percent), methadone/buprenorphine (0.2 percent) and other opiates/opioids (0.4 percent) for a non-medical, non-prescribed use.

**Source:** AIHW (2014).

Substance use disorder is diagnosed if substance use causes clinically and functionally significant impairment. The DSM-IV-TR included two broad categories: substance abuse and substance dependence. However, problems with these two categories, including low reliability and results of studies

suggesting that the criteria fit better in one category rather than in two, led to revisions in DSM-5 (Hasin et al., 2013). The DSM-5 has just one category: **substance use disorder**. The differences between DSM-IV-TR and DSM-5 are depicted in figure 7.1.



The DSM-5 contains substance use disorder categories for specific substances, such as alcohol, opioids and tobacco. New to DSM-5 is the inclusion of gambling disorder in the chapter on substance-related and addictive disorders. Table 7.2 lists the DSM-5 severity ratings for substance use disorders. In the DSM-5, meeting six or more of the diagnostic criteria is considered severe substance use disorder.

**TABLE 7.2** Severity ratings for substance use disorders in DSM-5

Rating number of diagnostic	Criteria met
Mild	2–3 criteria
Moderate	4–5 criteria
Severe	6 or more criteria

Substance use disorder includes two physiological symptoms: tolerance and withdrawal. **Tolerance** is indicated by either (1) larger doses of the substance being needed to produce the desired effect or (2) the effects of the drug becoming markedly less if the usual amount is taken. **Withdrawal** refers to the negative physical and psychological effects that develop when a person stops taking the substance or reduces the amount. Substance withdrawal symptoms can include muscle pains and twitching, sweats, vomiting, diarrhoea and insomnia. A separate withdrawal syndrome is defined for each substance in the DSM-5.

The most recent statistics on the prevalence of substance use disorders in the Australian population come from the 2007 Australian National Survey of Mental Health and Wellbeing (NSMHWB). This survey collected information on the DSM-IV-TR substance use disorders (dependence and abuse). More recent Australian data on the prevalence of the DSM-5 substance use disorder has not been collected. According to the NSMHWB, 5.1 percent of Australians over the age of 16 met criteria for a substance use disorder in the previous 12 months (Slade, Johnston, Oakley Browne, Andrews, & Whiteford, 2009). Alcohol use disorders were the most prevalent, with 2.9 percent of the Australian population over the age of 16 meeting criteria for alcohol abuse and 1.4 percent meeting criteria for substance dependence (Teesson et al., 2010).

Drug and alcohol use disorders are among the most stigmatised of disorders. Terms such as *addict* or *alcoholic* are tossed about carelessly, as if these words capture the essence of people, not the disorder from which they suffer. Historically, drug and alcohol problems have been viewed as moral lapses or a lack of willpower rather than as conditions in need of treatment. Unfortunately, such attitudes persist today. While people make decisions about whether or not to try alcohol or drugs, the ways in which

these decisions and the substances involved interact with an individual's neurobiology, social setting, culture and other environmental factors all conspire to create a substance use disorder. Thus, it is a mistake to consider substance use disorders as somehow being the result of moral failing, personal choice or lack of willpower.

We turn now to an overview of some of the major substance use disorders, those involving alcohol, tobacco, cannabis, opiates and stimulants.

#### CLINICAL CASE

##### Costa

Costa was 54 years old and living alone when his family finally persuaded him to talk to his general practitioner about his alcohol use. He had taken a bad fall while drunk and it may have been this event that finally got him to admit that something was wrong. He felt his drinking had been out of control for several years. He began each day with a drink, continued through the morning and was totally intoxicated by the afternoon. He seldom had any memory for events after noon of any day. Since early adulthood he had drunk regularly, but rarely during the day and never to the point of being drunk. The sudden death of his wife in a car accident two years earlier had triggered a quick increase in his drinking and within six months he had slipped into a pattern of heavy drinking. He had little desire to go out of his house and had cut back on social activities with family and friends. Repeated efforts by his family to get him to curtail his intake of alcohol had only led to angry confrontations.

His GP did a medical examination and analysed his liver enzymes. While Costa's liver was not enlarged, a number of liver enzymes were elevated. The GP referred Costa to a clinical psychologist for cognitive-behavioural therapy to address his alcohol use.

##### QUESTIONS

1. Which of the following characterise alcohol and drug use dependence?
  - (a) A strong desire to take alcohol or drugs
  - (b) Difficulty in controlling use
  - (c) Health, behavioural and social problems attributed to drug and alcohol use
  - (d) All of the above
2. List the health, behavioural and social problems attributed to drug and alcohol use in the case of Costa.

## Alcohol use disorder

The use of alcohol is a serious public health concern and leads to considerable burden to individuals and society. Data from the 2013 Australian National Household Drug Survey indicates that 78.2 percent of Australians aged over 14 years have used alcohol recently, with 18.2 percent of Australians drinking at levels that put them at risk for lifetime harms (AIHW, 2014). Alcohol use contributes heavily to the burden of disease in the Australian population, with 5.1 percent of total disease burden attributable to alcohol use (AIHW, 2016b). In younger Australians (up to the age of 44 years), alcohol use is the number one risk factor contributing to total disease burden. In all other age groups, alcohol use ranks within the top 10 risk factors contributing to disease burden.

### Prevalence of alcohol use disorder

Results from the 2007 NSMHWB indicated that 4.3 percent of the Australian population over the age of 16 met the criteria for the DSM-IV-TR categories of alcohol dependence or alcohol abuse in the previous 12 months (Teesson et al., 2010). The diagnosis of alcohol use disorder in DSM-5 is relatively new, so we do not yet have Australian prevalence estimates for this category.

Alcohol use disorders are especially frequent among young adults in the Australian population. Data from the 2007 NSMHWB indicated that 11.1 percent of young adults (aged 16–24 years) met criteria for alcohol abuse or dependence in the previous 12 months (Mewton, Teesson, Slade, & Grove, 2011). When compared with their older counterparts, young adults are also more likely to drink at levels that put them at risk for injury on a single occasion of drinking, defined, as noted in table 7.3, as having four or more drinks on a single occasion (AIHW, 2014). Australians in their teens and 20s are also more likely to consume 11 or more standard drinks in a single occasion, putting them at very high risk for short-term injury. Compared with all other age groups, people in their 40s, however, are more likely to drink at levels that put them at risk of alcohol-related harm over their lifetime, defined as consuming more than two standard drinks per day on table 7.3 outlines the Australian National Health and Medical Research Council alcohol consumption guidelines.

**TABLE 7.3** Australian National Health and Medical Research Council alcohol guidelines

Guidelines	
Reducing the risk of alcohol-related harm over a lifetime	For healthy men and women, drinking no more than two standard drinks on any day reduces the lifetime risk of harm from alcohol-related disease or injury.
Reducing the risk of injury on a single drinking occasion	For healthy men and women, drinking no more than four standard drinks on a single occasion reduces the risk of alcohol-related injury arising from that occasion.
Children and young people under the age of 18	A. Parents and carers should be advised that children under 15 years of age are at the greatest risk of harm from drinking and that for this age group, not drinking alcohol is especially important. B. For young people aged 15–17 years, the safest option is to delay the initiation of drinking for as long as possible.
Pregnancy and breastfeeding	A. For women who are pregnant or planning a pregnancy, not drinking is the safest option. B. For women who are breastfeeding, not drinking is the safest option.

In general, more men than women have problems with alcohol, though this varies by age. In the 2007 NSMHWB, 5.9 percent of men met the criteria for alcohol use disorders compared with 2.7 percent of women (Teesson et al., 2010). When only focusing on those aged 16–24, however, 13.1 percent of males and 8.9 percent of females met criteria for an alcohol use disorder (Mewton et al., 2011). Males are also consistently more likely than females to drink at levels that put them at single occasion risk of injury and lifetime risk of harm (AIHW, 2014). International evidence also indicates that the gap between men and women is closing in terms of alcohol use and related harms. In a review of 68 studies around the globe, it was found that in the early 1900s men were twice as likely as women to consume alcohol and three times as likely to experience alcohol-related problems (Slade et al., 2016). However, in more recent cohorts the rates of consumption and alcohol-related problems were nearly equalised among men and women. These findings suggest that young women in particular should be the targets of prevention and intervention efforts to reduce alcohol use and harms, which have traditionally focused on males.

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Young women in particular should be the target of prevention and intervention efforts to reduce alcohol use and harms as these efforts have been traditionally focused on males, despite the rate of consumption and alcohol-related problems being nearly equal between men and women.



The prevalence of alcohol problems differs by ethnicity and country of birth as well. When compared to people born in a non-English-speaking country, those born in Australia and other English-speaking countries are over three times as likely to meet criteria for an alcohol use disorder (Teesson et al., 2010). Indigenous Australians are more likely than their non-Indigenous counterparts to abstain from alcohol (AIHW, 2014). However, those who do consume alcohol are more likely to drink at levels that put them at risk for short-term injury and long-term harm.

Alcohol use disorders are highly comorbid with mental and other substance use disorders. According to the 2007 NSMHWB, 20 percent of people with an alcohol use disorder also met criteria for another disorder, compared with 8 percent of the rest of the sample (Teesson et al., 2010). When compared with the rest of the population, those with an alcohol use disorder were nearly 20 times as likely to meet criteria for a comorbid substance use disorder and over two and a half times more likely to meet criteria for a comorbid anxiety disorder. The rates of affective disorders, however, were similar among those who had met criteria for an alcohol use disorder and those who hadn't.

### **Short-term effects of alcohol**

How does alcohol produce its short-term effects? After being swallowed and reaching the stomach, alcohol begins to be metabolised by enzymes. Most of it goes into the small intestine and from there it is absorbed into the blood. It is then broken down, primarily in the liver. The effects of alcohol vary with its concentration in the bloodstream. Levels in the bloodstream depend on the amount ingested in a particular period of time, the presence of food in the stomach (food retains the alcohol and reduces its absorption rate), the weight and body fat of the person drinking, the efficiency of the liver and genetics. The same amount of alcohol will thus have a different effect on a 90 kilogram man who has just eaten than on a 50 kilogram woman with an empty stomach. However, women achieve higher blood alcohol concentrations even after adjustment for differences in body weight, perhaps due to differences in body water content between men and women.

It is also important to consider the question: what counts as a standard drink? A standard drink is defined by the Australian National Health and Medical Research Council as 10 grams of pure alcohol. Because alcoholic beverages contain different amounts of pure alcohol, it is not the size of the drink

that matters but the alcohol content of the particular beverage. The number of standard drinks in various beverages and serving sizes can be found here: [www.nhmrc.gov.au/health-topics/alcohol-guidelines](http://www.nhmrc.gov.au/health-topics/alcohol-guidelines)

Alcohol produces its effects through its interactions with several neural systems in the brain. It stimulates gamma-aminobutyric acid (GABA) receptors, which may account for its ability to reduce tension. GABA is a major inhibitory neurotransmitter; the benzodiazepines, such as Xanax, have an effect on GABA receptors similar to that of alcohol. Alcohol also increases levels of serotonin and dopamine, which may be the source of its ability to produce pleasurable effects. Finally, alcohol inhibits glutamate receptors, which may cause the cognitive effects of alcohol intoxication, such as slowed thinking and memory loss.

### Long-term effects of prolonged alcohol abuse

Almost every tissue and organ of the body is adversely affected by prolonged consumption of alcohol. For example, alcohol impairs the digestion of food and absorption of vitamins. In older people who have chronically consumed alcohol at harmful levels, a deficiency of B-complex vitamins can cause amnesic syndrome, a severe loss of memory for both recent and long-past events. Heavy, chronic alcohol consumption also has an effect on the structure and the function of the brain, particularly in the frontal lobes, cerebellum and limbic system (i.e., the amygdala, hippocampus and hypothalamus) (Oscar-Berman & Marinković, 2007).

Prolonged alcohol use plus reduction in the intake of proteins contributes to the development of cirrhosis of the liver, a disease in which some liver cells become engorged with fat and protein, impeding their function. Some cells die, triggering an inflammatory process and when scar tissue develops, blood flow is obstructed.

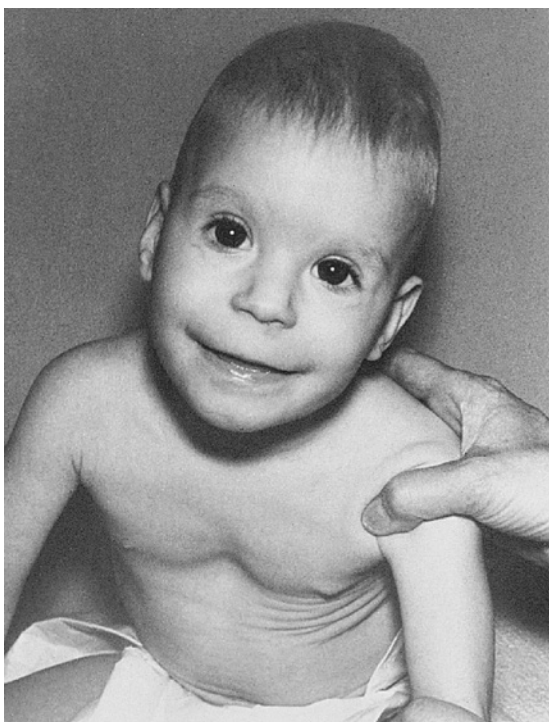
Other common changes to the body due to drinking include damage to the endocrine glands and pancreas, heart failure, erectile dysfunction, hypertension, stroke and capillary haemorrhages, which are responsible for the swelling and redness in the face, especially the nose, of people who chronically use alcohol at high levels.

Heavy alcohol consumption by a woman during pregnancy can lead to foetal alcohol spectrum disorders (FASD), including **foetal alcohol syndrome (FAS)**, partial foetal alcohol syndrome (pFAS), alcohol-related birth defects (ARBD) and alcohol-related neurodevelopmental disorder (ARND) (Burns, Breen, Bower, O'Leary, & Elliott, 2013). In FASD the growth of the foetus is slowed, and cranial, facial and limb anomalies can be produced, as well as behavioural and emotional problems. There is very little information on the prevalence of FASD in the Australian population, although one study suggested that the birth prevalence of FAS and pFAS was 0.06/1000 live births in Australia in the period from 2001 to 2004 (Elliott, Payne, Morris, Haan, & Bower, 2008). Even moderate drinking can produce undesirable, if less severe, effects on the foetus, leading the Australian National Health and Medical Research Council to advise abstinence during pregnancy as the safest course.

While heavy alcohol consumption has been linked with more than 200 acute and chronic conditions, there is some controversy regarding the potential positive effects associated with light

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Heavy drinking during pregnancy can cause foetal alcohol syndrome. Children with this disorder can have facial abnormalities and intellectual developmental disorder.



drinking. The relationship between alcohol consumption and mortality is popularly thought to follow a J-shaped curve, suggesting light consumption confers benefits when compared with both abstinence and heavy drinking. Light drinking, for example, has been related to lower risk for coronary heart disease and stroke (Kloner & Rezkalla, 2007; Sacco et al., 1999; Theobald, Bygren, Carstensen, & Engfeldt, 2000). It is argued that the positive effects of light alcohol consumption could be due to physiological (e.g., acetate, a metabolite of alcohol, increases coronary blood flow) or psychological (a less-driven lifestyle and diminished hostility) factors or, most likely, the interaction between the two factors. This popular belief, however, has recently been challenged by a study that looked more closely at the group of people classified as abstainers (Knott, Coombs, Stamatakis, & Biddulph, 2015). While typical studies tend to include both former drinkers and never drinkers in the abstainer group, this study excluded former drinkers who may be abstaining due to alcohol-related health issues or poor health in general. When light drinkers were compared only with abstainers who had never consumed alcohol, there was little evidence of the protective effects of alcohol consumption even at low levels.

## Tobacco use disorder

**Nicotine** is the addicting agent of tobacco, and tobacco is one of the most widely used psychoactive substances in the general population. Over the past century, evidence has accumulated on the serious adverse effects of tobacco-smoking on the health of smokers as well as the adverse effects of involuntary exposure to tobacco on non-smokers.

### Prevalence and health consequences of smoking

Smoking is recognised as one of the largest preventable causes of death and disease in Australia, associated with the death of approximately 15 000 Australians each year. The rates of daily smoking among Australians aged 18 or older have nearly halved in the past 20 years, reducing from 26.1 percent in 1993 to 13.3 percent in 2013 (AIHW, 2014). Nevertheless, 2.5 million Australians were smoking daily in 2013 and tobacco use remains the leading risk factor contributing to burden of disease in the Australian population among those aged over 45 years (AIHW, 2016b). It is estimated that half of all smokers will die prematurely because they smoked (Intergovernmental Committee on Drugs, 2012).

Tobacco smoke is a complex mixture of more than 4000 chemicals in the form of gases, particles or both, many of which are harmful. Among the other medical problems associated with, and almost certainly caused or exacerbated by, long-term cigarette smoking are chronic obstructive pulmonary disease; cancers of the lung, larynx, oesophagus, pancreas, bladder, cervix, kidney and stomach; Parkinson's disease; Crohn's disease; ulcerative colitis; asthma; macular degeneration; otitis media; complications during pregnancy (low birth weight, antepartum haemorrhage); sudden infant death syndrome; and a number of cardiovascular disorders (Lopez, 2006).

California's Tobacco Education Media Campaign parodies tobacco ads to illustrate health risks associated with smoking and to attack pro-tobacco influences.



In 2013, females were less likely to smoke than males and far fewer younger people were smokers compared with older age groups (AIHW, 2014). Only 3.4 percent of adolescents aged 12–17 years smoked tobacco daily. Among males, those aged 40–49 years were the most likely to smoke, whereas for females, those aged 25–29 years were most likely to smoke. There is also evidence for a delay in the uptake of smoking, with the average age at which young people smoked their first full cigarette rising steadily between 1995 (14.2 years) and 2013 (15.9 years). Australian data also indicate that the average number of cigarettes smoked per week among current smokers is also declining. In 2013, one in five smokers had successfully given up smoking for at least a month and three in 10 smokers had tried to quit but did not succeed. People living in areas with the lowest socioeconomic status (SES) were three times more likely to smoke than people with the highest SES.

Research demonstrates the significance of ethnicity in nicotine addiction. While the rates of daily smoking reduced among those who speak English at home between 2010 and 2013, the rates increased among those people who speak a language other than English in the same time period (AIHW, 2014). Rates of tobacco smoking have been identified as particularly high among men of Chinese, Arab and Vietnamese backgrounds. Indigenous Australians are more likely to die of smoking-related illnesses. In 2013, Indigenous Australians were 2.5 times as likely to smoke daily when compared with their non-Indigenous counterparts (32 percent compared with 12.4 percent). Unlike their non-Indigenous counterparts, the rates of daily smoking did not decline among Indigenous Australians between 2010 and 2013.

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Parental smoking greatly increases the chances that children will begin to smoke.



### Health consequences of secondhand smoke

As we have known for many years, the health hazards of smoking are not restricted to those who smoke. The smoke coming from the burning end of a cigarette, so-called **secondhand (or passive) smoke**, contains higher concentrations of ammonia, carbon monoxide, nicotine and tar than does the smoke actually inhaled by the smoker. The effects of secondhand smoke include the following.

- Non-smokers can suffer lung damage, possibly permanent, from extended exposure to cigarette smoke. Those living with smokers are at greatest risk. Precancerous lung abnormalities have been observed in

those living with smokers, and non-smokers are at greater risk for developing cardiovascular disease and lung cancer. In addition, some non-smokers have allergic reactions to the smoke from burning tobacco.

- Babies of women exposed to secondhand smoke during pregnancy are more likely to be born prematurely, to have lower birth weights and to have birth defects.
- Children of smokers are more likely to have upper respiratory infections, asthma, bronchitis and inner-ear infections than are their peers whose parents do not smoke. Secondhand smoke can cause sudden infant death syndrome (SIDS).

The best form of prevention for exposure to secondhand smoke is to promote smoke-free environments. Measures towards creating smoke-free environments have been established nationally and within the separate Australian states and territories. Across the country, smoking is banned in enclosed public places. There is, however, some variability between states and territories in terms of exemptions from indoor bans. Different jurisdictions also have different approaches for managing smoking in outdoor areas. Across Australia, the proportion of households with dependent children where someone smoked inside the home decreased from 31 percent in 1995 to 3.7 percent in 2013 (AIHW, 2014).

### E-cigarettes

Electronic cigarettes or e-cigarettes look like cigarettes except that they are made of plastic or metal and are filled with liquid nicotine that is mixed with other chemicals and often with flavours. They are battery operated (hence the 'e' for electronic) and work by heating up a nicotine liquid concoction so that people inhale and exhale the vapour. The term *vaping* has come to mean smoking an e-cigarette. Some models include a light at the end of the tube to mimic a lit cigarette. These are marketed as safe alternatives to cigarettes because they do not contain the tar and carbon monoxide that come from burning cigarettes. Although it is true that these devices do not have to contain nicotine (many are sold with flavoured water vapour), they can all be used to deliver liquid nicotine to users.

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E-cigarettes are becoming more popular even though we do not yet know about the safety of these products.



E-cigarette use has grown rapidly in parts of the world, particularly in the United Kingdom and United States. However, the prevalence of e-cigarette use in Australia is not well documented. In 2013, 14.8 percent of smokers aged 14 years or older had used an e-cigarette in the previous 12 months, with younger smokers more likely to have used an e-cigarette than older smokers (AIHW, 2014). Male smokers were also more likely than female smokers to have used an e-cigarette.

Some argue that e-cigarettes are safer alternatives to cigarettes containing tar and other carcinogens and that they may assist people who want to quit smoking actual cigarettes, a topic we return to later in the chapter. Others argue that nicotine is still an extremely addictive drug. Because they are so new, very little research has been done on the safety of these products, although several studies are currently underway. The laws relating to e-cigarettes in Australia are complex and vary by state and territory. As of October 2016, the sale and personal possession of e-cigarettes containing nicotine is unlawful across Australia. If the e-cigarette is to be used for therapeutic purposes (such as smoking cessation) it would need to be registered with the Therapeutic Goods Administration (TGA) in order to be sold lawfully and could only be purchased with a prescription. However, there are currently no e-cigarette products containing nicotine that are registered with the TGA. E-cigarettes may be imported for personal use, however, as these are exempt from TGA requirements. A prescription from a medical practitioner is still required to import e-cigarettes for personal use.

## Cannabis

**Cannabis** consists of the dried and crushed leaves and flowering tops of the hemp plant, *Cannabis sativa*. It is most often smoked, but it may be chewed, prepared as a tea or eaten in baked goods. **Hashish**, much stronger than cannabis, is produced by removing and drying the resin exudate of the tops of cannabis plants. In DSM-5, cannabis use disorder is the category name that includes cannabis-related problems.

Synthetic cannabis contains artificially created chemicals similar to those contained in cannabis. These chemicals are typically sprayed onto inert plant materials and placed in small packages and sold under names such as Spice or K2. Synthetic cannabis was made illegal in 2011. In 2013, 1.3 percent of the population had used synthetic cannabis in the previous 12 months, with those aged 14–19 years and 20–29 years the most likely age groups to have used synthetic cannabis (2.8 percent and 2.5 percent respectively) (AIHW, 2014). Little is known about synthetic drugs in general because they are so new; they will be the focus of much future research given their popularity among young people and concerns about their effects.

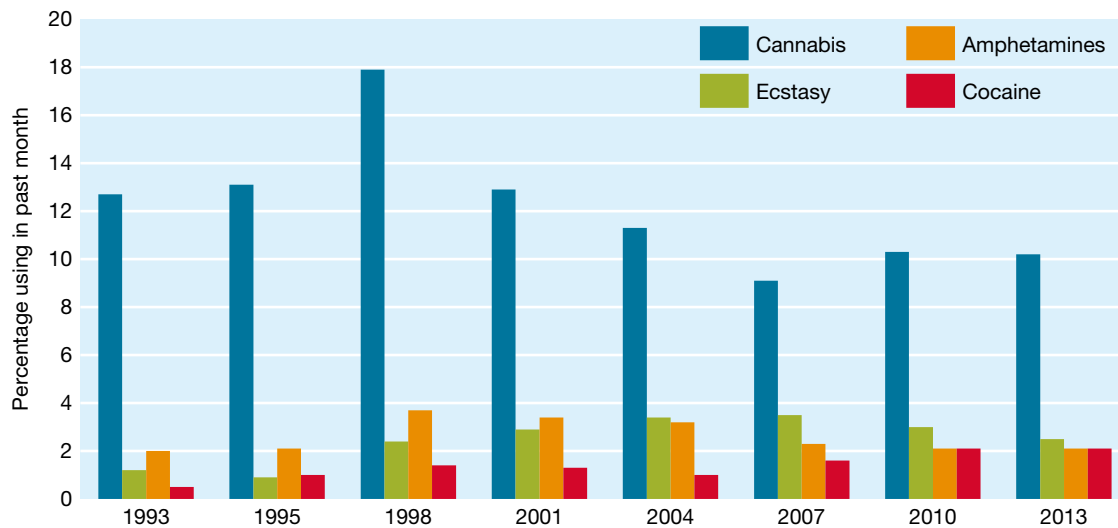
### Prevalence of cannabis use

Cannabis is the most frequently used illicit drug. In Australia in 2013, 6.6 million people (or 35 percent of people) aged 14 or older reported using cannabis in their lifetime and about 1.9 million (or 10.2 percent) had used cannabis in the previous 12 months (AIHW, 2014). Cannabis is the most commonly used illicit drug across all age groups (see figure 7.2 for data on usage among Australians aged 14 years or older). The prevalence of cannabis use in the previous 12 months is higher among men than women (12.8 percent versus 7.6 percent). Indigenous Australians are nearly twice as likely to have used cannabis in the previous 12 months when compared with non-Indigenous people.

### Effects of cannabis

Cannabis is a complex drug that comprises approximately 80 unique cannabinoids along with many other compounds. The major psychoactive chemical in cannabis is delta-9-tetrahydrocannabinol (THC). The amount of THC in cannabis, and thus its potency, is variable. Due to a lack of longitudinal data, it is not currently known whether the potency of cannabis has increased in Australia, although potency has been shown to be increasing internationally (McLaren, Swift, Dillon, & Allsop, 2008).

**FIGURE 7.2** Trends in past month drug use among young people aged over 14



*Source:* Based on data from AIHW (2014).

### Psychological effects

The intoxicating effects of cannabis, like those of most drugs, depend in part on the potency and size of the dose. Smokers of cannabis find that it makes them feel relaxed and sociable. Large doses have been reported to bring rapid shifts in emotion, to dull attention, to fragment thoughts, to impair memory and to give the sense that time is moving more slowly. Extremely heavy doses have sometimes been found to induce hallucinations and other effects similar to those of hallucinogens, including extreme panic, sometimes arising from the belief that a frightening experience will never end. Dosage can be difficult to regulate because it may take up to half an hour after smoking cannabis for its effects to appear; many users thus get much higher than intended.

### RESEARCH EXAMPLE

#### Is cannabis a gateway drug?

The so-called stepping-stone, or gateway, theory of cannabis use has been around for a long time. According to this view, cannabis is dangerous not only in itself but also because it is a first step for young people on the path to becoming addicted to other drugs, such as heroin.

Is there evidence that cannabis is indeed a gateway to more serious substance use? Overall, the evidence suggests that the answer to this question is no. For example, about 40 percent of weekly cannabis users do *not* go on to use other illicit substances (Degenhardt et al., 2010). So if by *gateway* we mean that escalation to a more serious drug is inevitable, then cannabis is not a gateway drug. However, we do know that many, but far from all, who use heroin and cocaine began their drug experimentation with cannabis and that cannabis users are more likely than non-users to experiment later with heroin and cocaine (Degenhardt et al., 2010).

Thus, even though cannabis use often precedes other drug use, it does not appear to *cause* later drug use, as the term *gateway* implies. Rather, it may be that cannabis is the first drug to be tried because it is more socially acceptable than other drugs. Time will tell whether legalisation of cannabis will increase the likelihood of other serious drug use, as some critics of legalisation argue, or will instead be more like cigarettes and alcohol — legal drugs that are associated with other drug use but not necessarily the gateway to their use.

Most people who use cannabis do not go on to use heroin, but many heroin users do begin their drug use with cannabis.



### QUESTIONS

1. Is there evidence that cannabis is indeed a gateway to more serious substance use?
2. Should parents be worried that cannabis use in teenage years will lead to so-called harder drugs like heroin?

Accumulated scientific evidence indicates that cannabis causes acute cognitive impairments in learning, memory, working memory and attention but it is less clear whether cannabis use is associated with enduring cognitive impairments (Volkow et al., 2016). One large longitudinal study of people from New Zealand assessed cannabis use at five time points between the ages of 18 and 38. Neuropsychological functioning was assessed at age 13 prior to initiation of cannabis use and again at age 38. The researchers found that those people who persistently met the criteria for cannabis dependence (defined as the number of assessment periods out of five in which people met the diagnostic criteria for cannabis dependence) had decrements in IQ points and poorer performance on tests assessing working memory and processing compared to people who never used cannabis or did so less persistently (Meier et al., 2012). This was particularly true for those who began using cannabis chronically as adolescents. Those people in the study who used cannabis but not regularly showed no decline in IQ or impairment on the neuropsychological tests.

Studies have also demonstrated that the acute effects of cannabis can impair complex psychomotor skills necessary for driving. The latest review of the evidence reports that driving under the influence of cannabis appears to result in a two- to three-fold increase in the risk of motor vehicle crashes. In all jurisdictions in Australia, legislation has been passed or drafted that allows for roadside saliva testing for drug driving, including cannabis.

### **Physical consequences**

The short-term effects of cannabis include bloodshot and itchy eyes, dry mouth and throat, increased appetite, reduced pressure within the eye and somewhat raised blood pressure.

We know that the long-term use of cannabis can impair lung structure and function (Grinspoon & Bakalar, 1995). Even though cannabis users smoke far fewer cigarettes than do tobacco smokers, most inhale cannabis smoke more deeply and retain it in their lungs for much longer periods of time. Since cannabis has some of the same carcinogens found in tobacco, its harmful effects are greater than would be expected were only the absolute number of cigarettes or pipefuls considered. For example, one cannabis cigarette smoked in the typical way is the equivalent of five tobacco cigarettes in carbon monoxide intake, four in tar intake and 10 in terms of damage to cells lining the airways (Sussman, Stacy, Dent, Simon, & Johnson, 1996).

How does cannabis affect the brain? In the early 1990s, researchers identified two cannabinoid brain receptors, called CB1 and CB2 (Matsuda, Lolait, Brownstein, Young, & Bonner, 1990; Munro, Thomas, Abu-Shaar, 1993). CB1 receptors are found throughout the body and the brain, with a particularly high number in the hippocampus, an important region of the brain for learning and memory. Based on accumulating evidence, researchers have concluded that the cognitive problems associated with cannabis use are linked to the effects of cannabis on these receptors in the hippocampus (e.g., Sullivan, 2000).

In terms of the effect of cannabis on the structure and function of the brain, recent reviews have found fairly consistent evidence to suggest that cannabis use is associated with structural alterations in the amygdala and hippocampus, as well as the frontal and cerebellar regions of the brain. Impaired neural connectivity in the hippocampus and corpus callosum has also been identified in heavy cannabis users. Functional magnetic resonance imaging studies have also shown that cannabis users show altered neural activity in resting state and during cognitive testing (Volkow et al., 2016).

Is cannabis addictive? While historically cannabis has not been seen as a drug of dependence, cannabis dependence is now well recognised within the scientific community. One of the reasons that cannabis wasn't historically considered addictive was because there was no recognised cannabis withdrawal syndrome. The criteria for cannabis dependence in DSM-5 now include a cannabis withdrawal syndrome, which comprises the following: irritability, anger or aggression; nervousness or anxiety; sleep difficulty (e.g., insomnia, disturbing dreams); decreased appetite or weight loss; restlessness; depressed mood; physical symptoms causing significant discomfort including abdominal pain, shakiness/tremors, sweating, fever, chills or headache. According to the 2007 NSMHWB, about 1 percent of the Australian population met criteria for cannabis abuse or dependence and, of those who had ever used cannabis, 14.3 percent met criteria for abuse or dependence (Teesson et al., 2012).

### **Therapeutic effects and the legalisation of medical cannabis**

Research since the 1970s has established a number of therapeutic uses for cannabis. These include reduction in the nausea and loss of appetite that accompany chemotherapy for some people with cancer, glaucoma, chronic pain, muscle spasms, seizures and discomfort from AIDS.

Over the past 20 years, there has been increasing international focus on the potential of cannabis (or components of the drug) as a second-line treatment option for various medical conditions where the most effective medication is not working well. Since that time, 24 states across the United States have made cannabis available for medical purposes. In 2014–16, laws surrounding the medical use of cannabis also started changing in Australia. From November 2016, due to changes introduced by the TGA, medical cannabis will be able to be used on medical grounds across Australia, but the use or cultivation of cannabis for recreational purposes remains illegal. Legislation regarding the cultivation, sale and use of medical cannabis is now being drafted within each of the Australian jurisdictions. In New South Wales, for example, medical cannabis will be available for palliative care, whereas in Victoria medical cannabis will only be available for children with epilepsy.

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Demonstrators in Nimbin, Australia, advocate the legalisation of cannabis.



## Opiates

The **opiates** include opium and its derivatives morphine, **heroin** and codeine. This group of addictive drugs can, in moderate doses, relieve pain and induce sleep. Their abuse is coded in DSM-5 as opioid use disorder.

Opiates that can be legally prescribed as pain medications, including **oxycodone** and morphine, have also been used for non-medical purposes. Between 1992 and 2012 there was a 15-fold increase in the dispensing of pain medications listed on the Pharmaceutical Benefits Scheme (PBS) across Australia. Oxycodone was the main contributor to increased pain medication utilisation, with tramadol, buprenorphine and morphine also contributing to this rise (Blanch, Pearson, & Haber, 2014).

### Prevalence of opiate abuse and dependence

In Australia, heroin use peaked in the 1990s and then went down quickly after a sharp decrease in supply. There are enormous difficulties in gathering data, but the most recent Australian data indicates that 0.1 percent of the population used heroin in the previous 12 months and 1.2 percent of those aged over 14 years had used heroin in their lifetime (AIHW, 2014). The 2007 NSMHWB estimated that the 12-month prevalence of opioid dependence in the Australian population was 0.1 percent (Slade et al., 2009). Arguably more valid estimates extrapolated from heroin deaths, methadone clinic data and heroin-related arrests indicate that heroin dependence affects 6.9/1000 people aged 15–54 years (Hall, Ross, Lynskey, Law, & Degenhardt, 2000).

The most commonly used opiates are prescription pain medications that are taken for non-medical purposes. Approximately 600 000 people (or 3.3 percent of the Australian population over the age of 14) used pain medications for non-medical purposes in 2013 and this rate had increased from 2010 (AIHW, 2014). The number of people seeking treatment for dependence on pain medications trebled between 2002 and 2011 (Nielsen et al., 2015). One in five people in treatment for opioid dependence were being treated for dependence on pharmaceutical opioids — most commonly morphine, codeine, oxycodone and fentanyl. Treatment demand was greater in regional and rural areas. Women make up the majority of people in treatment for codeine dependence, although the number of men in treatment for codeine

dependence is increasing over time. In contrast, men were far more likely than women to be in treatment for strong painkillers such as oxycodone and fentanyl.

The illicit supply of pain medications comes from prescriptions that are forged, stolen or diverted to dealers on the black market. Initially, OxyContin came in a pill format with polymer coating that made it easy to dissolve or crush into a form that could then be injected or snorted. In 2010, an extended-release formula that is not as susceptible to crushing for injection replaced the polymer-coated pills. Recent studies document a decline in the numbers of people using and injecting OxyContin after reformulation, with little evidence of switching to other drugs as a result (Degenhardt et al., 2015).

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Heroin was synthesised from opium in 1874 and was soon added to a variety of medicines that could be purchased without prescription. This ad shows a teething remedy containing heroin. It probably worked.



### Psychological and physical effects

Opiates produce euphoria, drowsiness and sometimes a lack of coordination. Heroin and OxyContin also produce a 'rush', a feeling of warm, suffusing ecstasy immediately after an intravenous injection. The user sheds worries and fears and has great self-confidence for four to six hours. However, the user then experiences a severe let-down, bordering on stupor.

Opiates produce their effects by stimulating neural receptors of the body's own opioid system (the body naturally produces opioids, called endorphins and enkephalins). Heroin, for example, is converted into morphine in the brain and then binds to opioid receptors, which are located throughout the brain. Some evidence suggests that a link between these receptors and the dopamine system is responsible for opiates' pleasurable effects. However, evidence from animal studies suggests that opiates may achieve their pleasurable effects via their action in the area of the brain called the nucleus accumbens, perhaps independently from the dopamine system (Koob, Caine, Hyttia et al., 1999).

Withdrawal from heroin may begin within eight hours of the last injection, at least after high tolerance has built up. During the next few hours after withdrawal begins, the person typically experiences muscle pain, sneezing and sweating, becomes tearful, and yawns a great deal. The symptoms resemble those of influenza. Within 36 hours, the withdrawal symptoms become more severe. There may be uncontrollable muscle twitching, cramps, chills alternating with excessive flushing and sweating, and a rise in heart rate and blood pressure. The person is unable to sleep, vomits and has diarrhoea. These symptoms typically persist for about 72 hours and then diminish gradually over a 5- to 10-day period.

Heroin dependence is a remarkably persistent, and in many cases a lifelong, condition with a high mortality rate (Teesson et al., 2015). The burden of disease associated with heroin and other opiate dependence is higher than any other illicit drug. Heroin overdose deaths are in the top 20 causes of years of life lost for males in developed countries. In an 11-year follow-up of over 600 heroin-dependent people in Australia, about 10 percent had died, nearly 70 percent had experienced a non-fatal overdose, over 40 percent had attempted suicide and a quarter were still using heroin. Six distinctive trajectory patterns of heroin use over 11 years were identified, illustrating greater heterogeneity in heroin use over time than has previously been found by other studies. The largest trajectory group (21.7 percent of the total cohort) consistently demonstrated high levels of use over the study period, emphasising that, for some, heroin dependence is a chronic, debilitating disorder requiring a long-term response (Darke et al., 2014; Teesson et al., 2015, 2017).

Heroin dependence is also associated with high rates of psychological comorbidity including high rates of depression and post-traumatic stress disorder. Recent trials of treatments for these comorbid conditions have shown promising results (Mills et al., 2012; Ross et al., 2016).

#### CLINICAL CASE

##### James

James was a 27-year-old man who had been addicted to heroin for seven years. He first tried heroin during his teens. He had experienced sexual abuse over a number of years by an older male relative and had witnessed his best friend dying in a car accident. After stealing money and valuables from his family to support his habit, he was asked to leave the house. He then began living on the street, dealing to support his habit. Over the years, James lost a tremendous amount of weight and became quite malnourished. Food wasn't a priority on most days, though he was usually able to gather a meal at the local men's hostel. James had been admitted to rehabilitation programs over the years, but returned to drug use within a few months of finishing treatment. More recently, James's sister had helped him to enter a methadone program. James tried methadone for a few weeks but was increasingly discouraged by the long waits outside the clinic each morning and the shame of being stared at by people passing on their way to work. The clinical psychologist at the methadone clinic assessed James for post-traumatic stress disorder and started a new program that provided prolonged exposure for PTSD in substance users (Back et al., 2014a, 2014b; Mills et al., 2012). James responded well to the treatment for PTSD and managed to stay on methadone, moving to collecting it from his local pharmacist. He was able to get job training and was hopeful for the future.

##### QUESTIONS

1. What are the common psychological comorbidities associated with heroin use?
2. Are there effective psychological and pharmacological responses to heroin dependence?

An additional problem associated with intravenous drug use is exposure, through sharing needles, to infectious agents such as the human immunodeficiency virus (HIV), which causes AIDS and hepatitis C. Notably, there is good consensus among scientists that needle exchange programs reduce needle sharing and the spread of infectious agents associated with intravenous drug use (Gibson, 2001; Yoast, Williams, Deitchman, & Champion, 2001). Needle exchange programs were introduced to Australia in 1987. Australian data indicate that these programs averted 32 050 new cases of HIV and 96 667 new cases of hepatitis C between 2000 and 2009 (National Centre in HIV Epidemiology and Clinical Research, 2009).

## Stimulants

**Stimulants** act on the brain and the sympathetic nervous system to increase alertness and motor activity. Amphetamines are synthetic stimulants; cocaine is a natural stimulant extracted from the coca leaf. Focus on discovery 7.1 discusses a less risky and more prevalently used stimulant, **caffeine**.

### FOCUS ON DISCOVERY 7.1

#### Our tastiest addiction — caffeine

What may be the world's most popular drug is seldom viewed as a drug at all and yet it has strong effects, produces tolerance in people and even subjects habitual users to withdrawal (Hughes, Higgins, Bickel, Hunt, & Fenwick, 1991). Users and non-users joke about it and most readers of this text have probably had some this very day. We are, of course, referring to caffeine, a substance found in coffee, tea, cocoa, cola and other soft drinks, some cold remedies and some diet pills.

Two cups of coffee, containing between 150 and 300 milligrams of caffeine, affect most people within half an hour. Metabolism, body temperature and blood pressure all increase; urine production goes up, as most of us will attest; there may be hand tremors, appetite can diminish and, most familiar of all, sleepiness is warded off. Extremely large doses of caffeine can cause headache, diarrhoea, nervousness, severe agitation, even convulsions and death. Death, though, is virtually impossible unless the person grossly overuses tablets containing caffeine, because the drug is excreted by the kidneys without any appreciable accumulation.

While it is assumed that caffeine improves mental alertness and performance, this may not be the case. One recent study investigated the effects of caffeine and caffeine withdrawal on mental alertness and performance among no/low coffee drinkers (<40 milligrams a day) and medium/high coffee drinkers ( $\geq 40$  milligrams a day) (Rogers, Heatherley, Mullings, & Smith, 2013). Among medium/high coffee drinkers there was no net benefit from consuming caffeine. While there was an increase in alertness and performance among this group, this represented a return to a normal (i.e., a reversal of the effect of caffeine withdrawal) state of affairs rather than an enhancement to above the normal state. Meanwhile, caffeine did appear to reduce sleepiness in low/moderate coffee drinkers but this was offset by an increase in anxiety and jitteriness, also resulting in no net benefit of caffeine.

#### QUESTIONS

1. Does caffeine improve mental alertness and performance?
2. What are the effects of extremely large doses of caffeine?

## Amphetamines

Amphetamines such as benzedrine, dexedrine and methedrine produce their effects by causing the release of norepinephrine and dopamine and blocking the reuptake of these neurotransmitters. **Amphetamines** are taken orally or intravenously and can be addicting. Wakefulness is heightened, intestinal functions are inhibited and appetite is reduced — hence their use in dieting. The heart rate quickens and blood vessels in the skin and mucous membranes constrict. The person becomes alert, euphoric and outgoing, and is possessed of seemingly boundless energy and self-confidence. Larger doses can make a person nervous, agitated and confused; other symptoms include palpitations, headaches, dizziness and sleeplessness. Sometimes heavy users become extremely suspicious and hostile, to the extent that they can be dangerous to others.

Tolerance to amphetamines develops rapidly, so more and more of the drug is required to produce the stimulating effect. One study has demonstrated tolerance after just six days of repeated use (Comer et al., 2001).

In Australia, 1.3 million people (7 percent) aged 14 years and over had used amphetamines in their life and 2.1 percent had used them in the previous 12 months (AIHW, 2014). Of those who had used amphetamines, 50.4 percent report crystal methamphetamine as the main form of the drug used.

## CLINICAL CASE

### Anton

Anton, a 37-year-old man, had just been arrested for breaking the conditions of his parole: stealing a package of cheese from a convenience store. He was also found to be under the influence of crystal methamphetamine (ice). Two months earlier, he had been released from prison after serving time for a break and enter, and for purchasing methamphetamine. He was determined to remain out of prison, but his cravings for methamphetamine were so intense that he was unable to abide by the terms of his parole. He had been using methamphetamine for two years on and off and had been arrested for drug-related offences, including prostitution (to get money to support his use).

One of Anton's friends says that Anton is a pretty intelligent guy, but the sad thing is how quickly his behaviour changes when he is on ice. He becomes aggressive and depressed in a matter of days... They say that they have experienced a lot of violence and aggression from Anton and that he previously never would have acted that way...

### QUESTIONS

1. What is the association between drug use and crime?
2. What are the behavioural consequences of crystal methamphetamine use?

### Methamphetamine

The most commonly abused stimulant drug is an amphetamine derivative called **methamphetamine**. Methamphetamine comes in three main forms: crystal (ice), base and powder (speed). Crystal and powder are the most frequently used forms and in 2013 crystal was more frequently used than powder, which had been the dominant form used in 2007 and 2010 (AIHW, 2014). Thus, while methamphetamine use as a whole hasn't been increasing in recent years in Australia, there has been a substantial increase in crystal methamphetamine use when compared to other forms of methamphetamine. Crystal usually has higher purity than powder forms and for this reason has stronger effects and lasts longer. It also has more potent side effects during use and in the 'comedown' or 'crash' phase. There is greater potential for methamphetamine users to develop dependence and psychosis, as well as long-term physical and mental health problems. There don't appear to be clear sex differences in terms of who uses methamphetamines. Among those aged 15–54 in Australia, it is estimated that 2.1 percent are regular users of methamphetamine and 1.2 percent are dependent users. Rates of regular and dependent use in Australia have been rising in the period 2002–14 (Degenhardt et al., 2016).

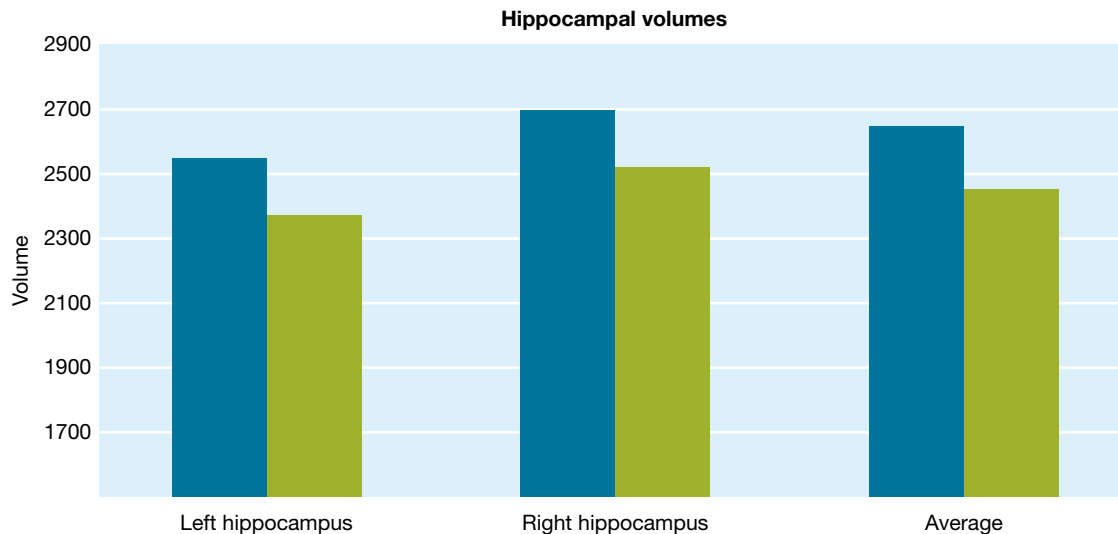
Craving for methamphetamine is particularly strong, often lasting several years after use is discontinued. Craving is also a reliable predictor of later use (Hartz, Frederick-Osborne, & Galloway, 2001). Methamphetamine takes effect quickly. The effects can last between 4 and 12 hours, although it can take one to two days for the drug to completely leave the body. The effects include feelings of euphoria and increased energy, as well as changes to the body, such as increased heart rate and blood pressure, dilated pupils, trembling, cramps and blurred vision. The effects eventually level off and then come crashing down. Not only do the good feelings crash, but the person also becomes very agitated. Apart from dependence, long-term effects of methamphetamine use also include dental problems; heart, kidney and lung problems; depression; paranoia; and psychosis.

Several animal studies have indicated that chronic use of methamphetamine causes damage to the brain, affecting both the dopamine and the serotonin systems (Frost & Cadet, 2000). Neuroimaging studies have found similar effects in the human brain, particularly in the dopamine system. For example, one study of chronic methamphetamine users found damage to the hippocampus (see figure 7.3). The volume of the hippocampus was smaller among chronic methamphetamine users, which correlated with poorer performance on a memory test (Thompson, Hayashi, Simon et al., 2004).

In a different study, men who were in treatment for methamphetamine dependence participated in a laboratory task of decision making while having their brains scanned with fMRI (Paulus, Tapert, & Schuckit, 2005).

The researchers found that lower activation in several brain areas (dorsolateral prefrontal cortex, insula, and areas of the temporal and parietal lobes) during the decision-making task predicted relapse to methamphetamine abuse one year after treatment. It seems obvious that poor decision making might put one at higher risk for relapse. This study also showed that the brain areas that contribute to sound decision making are disrupted in some people who are dependent on methamphetamine. What is less clear is whether the methamphetamine damaged these areas or whether these areas were damaged before methamphetamine use began.

**FIGURE 7.3** Results from an fMRI study showing that those who abused methamphetamine (green bars) had smaller hippocampal volume (size) than those in the control group (blue bars) who did not abuse methamphetamine



Source: Adapted from Thompson et al. (2004).

A caveat should be noted here. One difficulty with conducting these types of studies is finding participants who use only the drug of interest (in this case, methamphetamine) so that any observed effects can be linked to that drug and not to others. However, it is difficult to find methamphetamine users who have not at some point used other substances, particularly alcohol and nicotine. For example, in one of the studies described earlier, the methamphetamine users did not differ from the control group in alcohol consumption, but they did smoke more (Thompson et al., 2004). Nevertheless, it seems clear that the deleterious effects of methamphetamine are many and serious.

## FOCUS ON DISCOVERY 7.2

### New psychoactive substances

*Emerging or new psychoactive substances* (NPS) is a term used to describe drugs that have mind-altering effects and are new to the market. In 2013, at least 348 new substances were detected (AIHW, 2014). NPS are designed specifically to mimic the effects of established illicit drugs and to circumvent existing drug laws. As such, NPS are often referred to as 'legal highs', although laws surrounding these drugs change and evolve quickly, with the importation, sale and consumption of NPS now illegal in Australia.

Synthetic cannabis (discussed previously) is one of the most common NPS, but others include synthetic cathinones (or 'bath salts', synthetic stimulants), phenethylamines (designed to mimic the effects of ecstasy and amphetamine) and the NBOMe series (modelled on hallucinogens). As NPS change frequently, very little is known about their effects and risks over the long term, although short-term side effects include psychosis, nausea and hyperventilation. The fast-paced nature in which NPS are emerging, in conjunction with the paucity of knowledge about their toxicology and effects and constant changes in laws regarding their use, make these drugs potentially very harmful.

To date, the prevalence of NPS use has not been documented well in Australia. The 2013 National Drug Strategy Household Survey (NDSHS) was the first to document the prevalence of NPS use across Australia (AIHW, 2014). As indicated above, 1.3 percent of the population had used synthetic cannabis in the past 12 months and 0.4 percent of the population had used another NPS. The rates of use were considerably higher among young people, with 2.5 percent of those aged 20–29 years having used synthetic cannabis in the last 12 months and 1.3 percent having used another NPS in the same timeframe. In another large study of Australian high school students (mean age 15 years), 3 percent reported ever having tried an NPS (Champion, Teesson, & Newton, 2015).

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### QUESTION

Emerging or new psychoactive substances (NPS) is a term used to describe which drugs?

## Cocaine

The drug **cocaine** comes from the leaves of the coca shrub. Cocaine comes in three main forms: a paste, which is often off-white or light brown; a powder, which is often white or off-white; or a white or off-white crystal rock known as crack cocaine. Cocaine can be sniffed (snorted), smoked in pipes or cigarettes, swallowed or even injected into the veins; some heroin users mix the two drugs. In Australia, cocaine is most commonly used in a powder form, which is often ingested by snorting. In 2013, 2.1 percent of Australians aged over 14 years had used cocaine in the previous 12 months (AIHW, 2014). Males were most likely to have used cocaine in the previous 12 months and use was highest among younger age groups.

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Women harvesting the leaves of a coca plant in Bolivia, South America. The leaves contain about 1 percent cocaine.



Cocaine acts rapidly on the brain, blocking the reuptake of dopamine in mesolimbic areas. Cocaine yields pleasurable states because dopamine left in the synapse facilitates neural transmission. Self-reports of pleasure induced by cocaine are strongly related to the extent to which cocaine has blocked dopamine reuptake (Volkow, Wang, Fischman, & Foltin, 1997). Cocaine can increase sexual desire and produce feelings of self-confidence, wellbeing and indefatigability. An overdose may bring on chills, nausea and insomnia, as well as strong paranoid feelings and terrifying hallucinations of insects crawling beneath the skin. Chronic use can lead to heightened irritability, impaired social relationships, paranoid thinking, and disturbances in eating and sleeping. Some, but not all, users develop tolerance to cocaine, requiring a larger dose to achieve the same effect. Other users may become more sensitive to cocaine's effects, which are believed to be a contributing factor in deaths after a fairly small dosage. Stopping cocaine use appears to cause severe withdrawal symptoms.

Cocaine is a vasoconstrictor, causing the blood vessels to narrow. As users take larger and larger doses of the purer forms of cocaine, they are more often rushed to emergency rooms and may die of an overdose, often from a heart attack. Cocaine also increases a person's risk for stroke and causes cognitive impairments, such as difficulty paying attention and remembering.

Because of its strong vasoconstricting properties, cocaine poses special dangers in pregnancy, for the blood supply to the developing foetus may be compromised. An MRI study compared the grey matter (i.e., neural tissue of the brain) volume of adolescents who were and were not exposed to cocaine prenatally. Adolescents who had been exposed to cocaine prenatally were found to have lower volumes in areas of the frontal cortex and nearby regions that aid in cognitive control and emotion regulation compared to adolescents who were not prenatally exposed to cocaine (Rando, Chaplin, Potenza, Mayes, & Sinha, 2013). However, the prenatally exposed adolescents were also more likely to have initiated their own substance use, so it is impossible to tell if the different brain sizes were the result of prenatal cocaine exposure or their own substance use. It is likely that both contributed.

## Other illicit drugs

### Hallucinogens

**Hallucinogens** (also known as psychedelics) are a category of drugs that cause perceptual distortions such as hallucinations. Hallucinations are experiences where people hear or see things that aren't really there or where perception is altered. For example, colours or shapes may appear to be changing, more brightly coloured or moving. Users may also experience unusual thoughts, feelings or beliefs. Unlike the hallucinations in schizophrenia, however, these are usually recognised as such by the person, who realises they are being caused by the drug. Hallucinogens can be naturally occurring or synthetic. The most commonly known synthetic hallucinogen is **LSD** (lysergic acid diethylamide), also known as acid, trips or tabs. Naturally occurring hallucinogens include magic mushrooms, DMT (dimethyltryptamine), mescaline (found in peyote) and salvia. Nearly 1 in 10 Australians over the age of 14 have used hallucinogens in their lifetime and 1.3 percent in the previous 12 months (AIHW, 2014).

In addition to hallucinations, hallucinogens can alter a person's sense of time (it seems to go slowly). A person using LSD may have sharp mood swings but can also experience an expanded consciousness such that he or she seems to appreciate sights and sounds like never before. Many users experience intense anxiety after taking LSD, in part because the perceptual experiences and hallucinations can provoke fears that they are 'going crazy'. For some, these anxieties unfold into full-blown panic attacks. The anxiety usually subsides as the drug is metabolised. **Flashbacks** are visual recurrences of perceptual experiences after the physiological effects of the drug have worn off. In DSM-5, the category 'hallucinogen persisting perception disorder' involves re-experiences of flashbacks and other perceptual symptoms that occurred during hallucinogen use, even though the drug is no longer used.

### Ecstasy

The hallucinogen-like substance **ecstasy** is made from **MDMA** (methylenedioxymethamphetamine). Not until the 1970s were the psychoactive properties of MDMA reported in the scientific literature. Ecstasy became illegal in Australia in 1987.

## CLINICAL CASE

### Tamara

Tamara tried ecstasy for the first time when she was at university. She went to her first rave and a friend gave her a pill. Within a short period of time, she began to feel almost magical, as if she was seeing everything around her in a new light. She felt incredibly close to her friends and even to men and women she had just met. Hugging and close dancing were intensely pleasurable, in a completely new way. She is finding she is spending a lot of her money on ecstasy and is taking a lot of time to get over the effects from the drugs. She hasn't made it to any Monday classes recently and is starting to fall behind.

### QUESTION

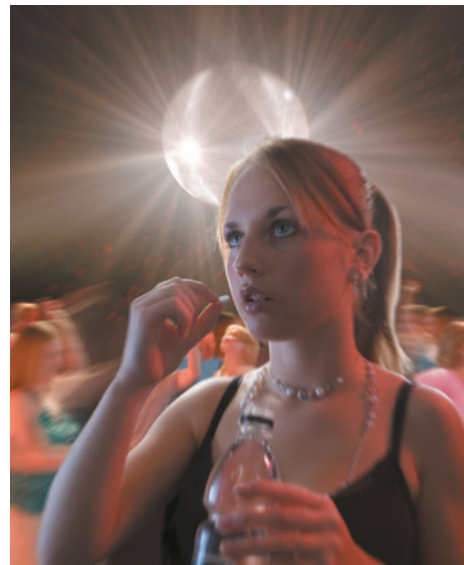
Describe some of the short-term effects of ecstasy use.

Ecstasy contains compounds from both the hallucinogen and amphetamine families, but its abuse is currently classified as 'other hallucinogen use disorder' in DSM-5. It can be taken in pill form, but MDMA is often mixed with other substances (e.g., caffeine) or drugs (e.g., LSD, ketamine, talcum powder), making the effects vary dramatically. After cannabis, ecstasy is the most commonly used illicit drug in a person's lifetime (10.9 percent of Australians over the age of 14 years) (AIHW, 2014). In 2013, 2.5 percent of Australian aged over 14 years reported using ecstasy in the previous 12 months. Ecstasy use peaked in the Australian population in 2007 before declining in 2010 and then again in 2013.

Ecstasy acts primarily by contributing to both the release and the subsequent reuptake of serotonin (Huether, Zhou, & Ruther, 1997; Liechti, Baumann, Gamma, & Vollenweider, 2000; Morgan, 2000), with recent reviews suggesting that it may have neurotoxic effects on the serotonin system (Roberts, Jones, & Montgomery, 2016; Vegting, Reneman, & Booij, 2016). It is difficult to say whether these toxic effects are directly due to drug use, since no studies in humans to date have assessed serotonin functioning both before and after ecstasy use.

Users report that ecstasy enhances intimacy and insight, improves interpersonal relationships, elevates mood and self-confidence and promotes aesthetic awareness. It can also cause muscle tension, rapid eye movements, jaw clenching, nausea, faintness, chills or sweating, anxiety, depression, depersonalisation and confusion.

Ecstasy is a popular party drug but, like many drugs, is not free of ill effects.



## FOCUS ON DISCOVERY 7.3

### Nitrous oxide

**Nitrous oxide** is a colourless gas that has been available since the nineteenth century. Within seconds of its ingestion, it induces lightheadedness and a state of euphoria in most people; for some, important insights seem to flood the mind. Many people find otherwise mundane events and thoughts irresistibly funny, hence its nickname, *laughing gas*.

Many people have received nitrous oxide at a dentist's office to facilitate relaxation and otherwise make a potentially uncomfortable and intimidating dental procedure more palatable. A major advantage of nitrous oxide over other analgesics and relaxants is that a person can return to a normal waking state within minutes of breathing enriched oxygen or normal air.

Sometimes called 'nangs', nitrous oxide balloons are often combined with the use of ecstasy and other drugs. According to the Australian Trends in Ecstasy and Related Drug Markets 2016 Survey, the rate of nitrous oxide use has increased significantly among Australians who also use ecstasy and related drugs from 2015 (25 percent of the sample) to 2016 (36 percent of the sample) (Stafford, Sutherland, Burns, & Breen 2016). Nitrous oxide fits in the broader category of inhalants and it has been illegal to knowingly or recklessly supply nitrous oxide since 2013.

Nitrous oxide is no laughing matter.



#### QUESTIONS

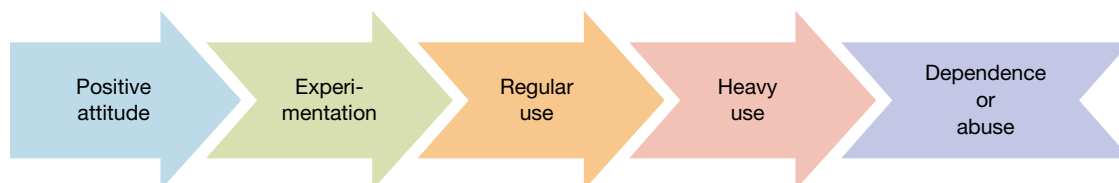
1. What is nitrous oxide?
2. What are some of the effects of using nitrous oxide?

## 7.2 Aetiology of substance use disorders

**LEARNING OUTCOME 7.2** Understand the major aetiological factors for substance use disorders, including genetic factors, neurobiological factors, mood and expectancy effects and sociocultural factors.

Becoming physiologically dependent on a substance is a developmental process for some people. That is, some people begin with a positive attitude towards a substance, then start to experiment with using it, then begin using it regularly, then use it heavily and finally become dependent on it (see figure 7.4).

**FIGURE 7.4** The process by which some people become dependent on a drug



Although applicable in many cases, the developmental process approach does not account for all cases of substance use disorders. For example, some people have periods of heavy use of a substance — for example, alcohol — and then return to moderate use. Other people do not need a period of heavy use to become dependent on the substance, as in the case of methamphetamine. In the following sections, we discuss genetic, neurobiological, psychological and sociocultural factors associated with substance use disorders. Keep in mind that these factors are likely to be differently related to different substances. Genetic factors, for example, may play a role in alcohol use disorder but may be less important in hallucinogen use disorder.

## Genetic factors

Much research has addressed the possibility that there is a genetic contribution to drug and alcohol use disorders. Several studies have shown that relatives and children of problem drinkers have higher-than-expected rates of alcohol use disorder (e.g., Chassin, Pitts, Delucia, & Todd, 1999). Stronger evidence for genetic factors comes from twin studies, which have revealed greater concordance in identical twins than in fraternal twins for alcohol use disorder (McGue, Pickens, & Svikis, 1992), smoking (True et al., 1999), heavy use of cannabis (Kendler & Prescott, 1998) and drug use disorders in general (Tsuang, Lyons, Meyer et al., 1998; Kendler, Myers, & Prescott, 2007). Other behavioural genetics studies indicate that the genetic and shared environmental risk factors for illicit drug use disorders may be rather non-specific (Kendler, Jacobsen, Prescott, & Neale, 2003). That is, genetic and shared environmental risk factors appear to be the same no matter what the drug (cannabis, cocaine, opiates, hallucinogens, sedatives, stimulants) and this appears to be true for both men and women (Kendler, Prescott, Myers, & Neale, 2003).

Of course, genes do their work via the environment, and research has uncovered gene–environment relationships in alcohol and drug use disorders (Kendler et al., 2012). Among adolescents, peers appear to be particularly important environmental variables. For example, a large twin study in Finland found that heritability for alcohol problems among adolescents was higher among those teens who had a large number of peers who drank compared to those who had a smaller number of peers who drank (Dick et al., 2007). The environment in this case was peer-group drinking behaviour. Another study found that heritability for both alcohol and smoking among adolescents was higher for those teens whose best friend also smoked and drank (Harden, Hill, Turkheimer, & Emery, 2008). In this case, the environment was best-friend behaviour. Another study found that heritability for smoking was greater for teens who went to schools where the ‘popular crowd’ smoked compared to schools where the popular students did not smoke (Boardman, Saint Onge, Haberstick, Timberlake, & Hewitt, 2008).

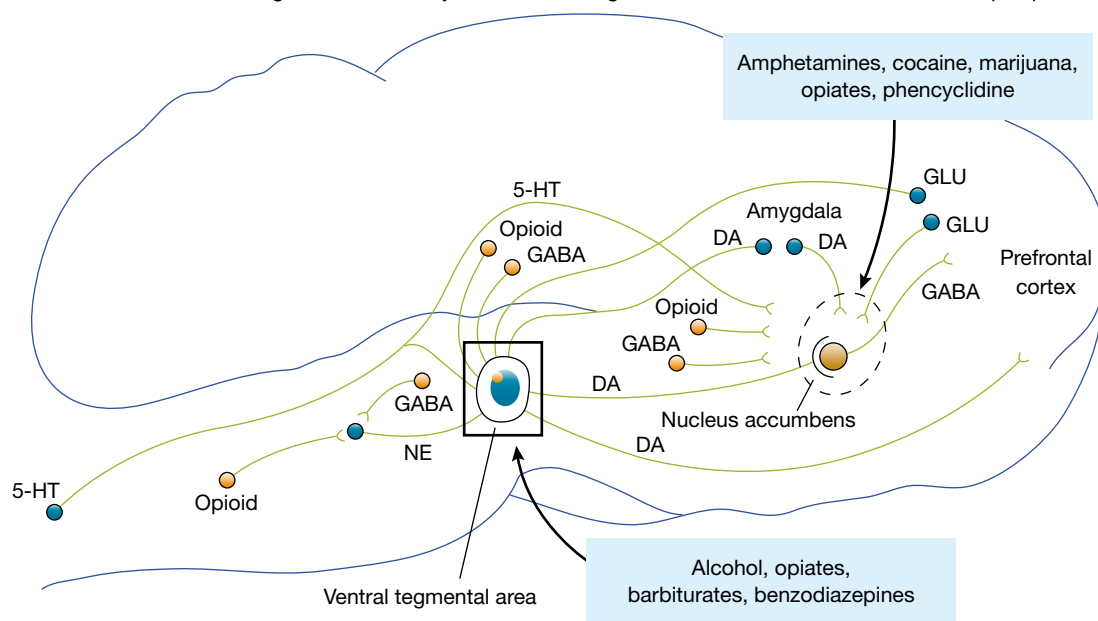
## Neurobiological factors

You may have noticed that in our discussions of specific drugs, the neurotransmitter dopamine has almost always been mentioned. This is not surprising given that dopamine pathways in the brain are linked to pleasure and reward. Drug use typically results in rewarding or pleasurable feelings and it is via the dopamine system that these feelings are produced. Research with both humans and animals shows that nearly all drugs, including alcohol, stimulate the dopamine systems in the brain (see figure 7.5), particularly the mesolimbic pathway (Camí & Farré, 2003; Koob, 2008). Researchers have wondered, then, whether problems in the dopamine pathways in the brain might somehow account for why certain people become dependent on drugs.

One of the difficult issues to resolve is whether problems in the dopamine system may increase the vulnerability of some people to becoming dependent on a substance, sometimes called the ‘vulnerability model’, or whether problems in the dopamine system are the consequence of taking substances (the ‘toxic effect model’). For drugs such as cocaine, there is currently research support for both views. Thus, this remains an important area to work out in future research.

**FIGURE 7.5**

Reward pathways in the brain that are affected by different drugs. DA = dopamine; GABA = gamma-aminobutyric acid; GLU = glutamate; 5-HT = serotonin; NE = norepinephrine



**Source:** Adapted from Camí & Farré (2003).

Although people take drugs to feel good, they also take them to feel less bad. This is particularly true once a person becomes dependent on a substance, such as alcohol, methamphetamine or heroin, whose withdrawal symptoms are excruciatingly unpleasant. In other words, people continue to take drugs to avoid the bad feelings associated with withdrawal. A substantial body of research with animals supports this motivation for drug-taking behaviour (Koob & Le Moal, 2008); this research helps to explain why relapse is so common.

## Cravings

Investigators have proposed a neurobiological theory, referred to as the *incentive-sensitisation theory*, which considers both the craving ('wanting') for drugs and the pleasure that comes with taking the drug ('liking') (Robinson & Berridge, 1993, 2003). According to this theory, the dopamine system linked to pleasure or liking becomes supersensitive not just to the direct effects of drugs but also to the cues associated with drugs (e.g., needles, spoons, rolling paper). This sensitivity to cues induces craving or wanting and people go to extreme lengths to seek out and obtain drugs. Over time, the liking for drugs decreases, but the wanting remains very intense. These investigators argue that the transition from liking to powerful wanting, accomplished by the drug's effects on brain pathways involving dopamine, is what maintains the addiction.

Many researchers study the neurobiology of wanting or craving. A number of laboratory studies have shown that cues for a particular drug can elicit responses not altogether unlike those associated with actual use of the drug. For example, those who were dependent on cocaine showed changes in physiological arousal, increases in cravings and 'high' feelings and increases in negative emotions in response to cues of cocaine, which consisted of audio and video recordings of people preparing to inject or snort cocaine, compared to people not dependent on cocaine (e.g., Robbins, Ehrman, Childress, Cornish, & O'Brien, 2000). Brain-imaging studies have shown that cues for a drug, such as a needle or a cigarette, activate the reward and pleasure areas of the brain implicated in drug use.

What about the psychology of craving? Do people who crave a substance more actually use it more, even if they are trying to quit? The answer appears to be yes. In a longitudinal study of heavy drinkers, the more participants reported wanting (craving) and liking samples of alcohol (carefully presented to

reduce expectancies about alcohol) at a baseline assessment, the more alcohol use disorder symptoms they had six years later (King, McNamara, Hasin, & Cao, 2014). Similarly, research has shown that pharmacotherapies used to treat alcohol dependence, such as naltrexone and acamprosate, have an effect on alcohol consumption by reducing craving (Richardson et al., 2008).

### **Valuing the short term over the long term**

A related psychological and neurobiological model emphasises the distinction between the value people place on short-term (immediate) versus long-term (delayed) rewards. People with a substance use disorder often value the immediate, even impulsive, pleasure and reward that comes from taking a drug more than delayed reward, such as a monthly pay check from work.

Laboratory experiments to assess whether people value immediate or delayed rewards typically present people with choices of monetary rewards that are immediate but small (e.g., \$1 now) or delayed but larger (e.g., \$10 in a day). The extent to which people opt for the smaller, immediate reward can be calculated mathematically and is often called delay discounting. In other words, researchers can compute the extent to which people discount the value of larger, delayed rewards. People who are dependent on alcohol and drugs, such as opiates, nicotine and cocaine, discount delayed rewards more steeply than do people not dependent on these substances (reviewed in Bickel, Koffarnus, Moody, & Wilson, 2014). One longitudinal study found that the extent of delay discounting predicted smoking initiation in adolescents that continued into early adulthood (Audrain-McGovern et al., 2009).

At the level of the brain, valuing immediate versus delayed rewards recruits different brain regions. Researchers have hypothesised that these brain regions compete with one another when people are faced with a decision of whether to take a drug. In fMRI studies, valuing the delayed reward is associated with activation of the prefrontal cortex; valuing the immediate reward is associated with activation in amygdala and nucleus accumbens (Bechara, 2005; Bickel et al., 2007).

## **Psychological factors**

In this section, we look at three other types of psychological factors that may contribute to the aetiology of substance use disorders. First, we consider the effects of drugs (particularly alcohol and nicotine) on mood; we examine the situations in which a tension-reducing effect occurs and the role of cognition in this process. Second, we consider people's expectancies about the effects of substances on behaviour, including beliefs about the prevalence with which a drug is used and about the health risks associated with using that drug. Third, we consider personality traits that may make it more likely for some people to use drugs heavily.

### **Mood alteration**

It is generally assumed that one of the main psychological motives for using drugs is to alter mood — that is, drug use is reinforced because it enhances positive moods or diminishes negative ones. For example, most people believe that an increase in tension (e.g., because of a bad day at work) leads to increased alcohol consumption.

Laboratory studies have demonstrated that alcohol reduces self-reported and physiological indicators of anxiety, particularly when there is uncertainty about a negative event (e.g., having arrived home late after having drinks with friends, will you argue with your spouse?) (Bradford, Shapiro, & Curtin, 2013). In addition, research has found that alcohol lessens negative emotions such as stress and anxiety, but it also lessens positive emotions in response to anxiety-provoking situations (Curtin, Lang, Patrick, & Strizke, 1998; Strizke, Patrick, & Lang, 1995). Longitudinal studies of stress and consumption have also provided support for this idea. For example, a longitudinal study of adolescent smokers found that increases in negative affect and negative life events were associated with increases in smoking (Wills, Sandy, & Yaeger, 2002). Other studies have found that life stress precedes alcohol-related relapses (e.g., Brady & Sonne, 1999).

Studies of the tension-reducing properties of nicotine have yielded mixed findings, with some studies showing that nicotine reduces tension and others not finding this effect (Kassel, Stroud, & Paronis, 2003). The reasons for the mixed findings may have to do with a failure to consider where people are in their

smoking behaviour. Did they just start smoking? Are they regular smokers? Have they tried to quit and failed? Research suggests that people experience a greater reduction of tension and negative affect when starting to smoke than when regularly smoking or when in relapse after treatment (Kassel et al., 2003; Shiffman & Waters, 2004). Why might this be? A laboratory study examined the types of situations associated with a reduction in negative affect after smoking (Perkins, Karelitz, Conklin, Sayette, & Giedgowd, 2010). Participants, who were regular smokers, had to give a speech, play a difficult computer game, abstain from smoking for 12 hours and view disturbing pictures. The researchers found that people experienced the greatest reduction in negative affect after the abstinence condition. That is, smoking, after not being able to smoke, provided more relief from negative affect than it did after other stressful situations; thus, the situation is important to consider when thinking about whether smoking reduces negative affect.

Other studies suggest that it is the sensory aspect of smoking (i.e., inhaling), not nicotine, that is associated with tension reduction. In the study just described, participants experienced a reduction in negative affect regardless of whether they were smoking cigarettes with or without nicotine (Perkins et al., 2010). Another experimental study randomly assigned smokers to have cigarettes with or without nicotine (indistinguishable by participants) after a negative or positive mood was induced (Perkins et al., 2008). The researchers also manipulated smokers' expectancies. That is, some smokers expected and received a cigarette with nicotine; others expected nicotine but didn't get it; others expected no nicotine and didn't get it; and others expected no nicotine but received it anyway. Smoking reduced negative affect after both mood inductions, but this was true for smokers regardless of what they expected and actually received to smoke (i.e., a cigarette with or without nicotine). Instead, the effects of inhaling, whether or not there was nicotine, had the greatest association with reducing negative affect.

Subsequent research to examine why substances appear to reduce tension in some situations but not in others has focused on situations in which distraction is present. These studies find that tension reduction is more likely to occur when distractions are present (Curtin et al., 1998; Josephs & Steele, 1990; Steele & Josephs, 1988). Why might this be? Alcohol impairs cognitive processing and narrows our attention to the most immediately available cues, resulting in 'alcohol myopia' (Steele & Josephs, 1990). In other words, an intoxicated person has less cognitive capacity and tends to use that capacity to focus on an immediate distraction, if available, rather than on tension-producing thoughts, with a resultant decrease in anxiety.

The benefits of distraction have also been documented for nicotine. Specifically, smokers who smoked in the midst of a distracting activity experienced a reduction in anxiety, whereas smokers who smoked without a distracting activity did not experience lessened anxiety (Kassel & Shiffman, 1997; Kassel & Unrod, 2000). However, alcohol and nicotine may increase tension when no distractions are present. For example, a person drinking alone may focus all his or her limited cognitive capacity on unpleasant thoughts, begin brooding and become increasingly tense and anxious, a situation reflected in the expression 'crying in one's beer'.

Tension reduction is only one aspect of the possible effects of drugs on mood. Some people may use drugs to reduce negative affect, whereas others may use them to increase positive affect when they are bored (Cooper, Frone, Russell, & Mudar, 1995). In this case, increased drug use results from a high need for stimulation combined with expectancies that drugs will promote increased positive affect. These patterns have been confirmed among people who abuse alcohol and cocaine (Cooper et al., 1995; Hussong, Hicks, Levy, & Curran, 2001).

### **Expectancies about alcohol and drug effects**

If substances don't always reduce stress and tension, why do so many people who drink or take drugs believe that it helps them unwind? Expectation may play a role here — that is, people may drink after stress not because it actually reduces tension but because they expect it to do so. In support of this idea, studies have shown that people who expect alcohol to reduce stress and anxiety are more likely to be frequent users (Rather, Goldman, Roehrich, & Brannick, 1992; Sher, Walitzer, Wood, & Brent, 1991; Tran, Haaga, & Chambless, 1997). Furthermore, the expectation that drinking will reduce anxiety increases drinking, which in turn makes the positive expectancies even stronger (Sher, Wood, Wood, & Raskin, 1996; Smith, Goldman, Greenbaum, & Christiansen, 1995).

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Expectations about alcohol influence whether people will drink.



Other research has shown that expectancies about a drug's effects — for example, the beliefs that a drug will stimulate aggression and increase sexual responsiveness — predict increased drug use in general (Stacy, Newcomb, & Bentler, 1991). Similarly, people who believe (falsely) that alcohol will make them seem more socially skilled are likely to drink more heavily than those who accurately perceive that alcohol can interfere with social interactions. In now-classic experiments demonstrating the power of expectancies, participants who believe they are consuming a quantity of alcohol when they are actually consuming an alcohol-free beverage subsequently become more aggressive (Lang, Goeckner, Adessor, & Marlatt, 1975). Alcohol consumption is associated with increased aggression, but expectancies about alcohol's effects can also play a role (Bushman & Cooper, 1990; Ito, Miller, & Pollock, 1996). Thus, as we have seen in other contexts, cognitions can have a powerful effect on behaviour.

### **Personality factors**

Personality factors that appear to be important in predicting the later onset of substance use disorders appear to be mediated by different reinforcement processes. Neurotic personality traits — for example, those related to anxiety and depression — appear to be linked to substance use through negative reinforcement motives. In such a pathway, an individual may use a substance to avoid feelings of depression or anxiety. Meanwhile, disinhibitory personality traits, such as impulsivity and sensation-seeking, appear to be linked to substance use through positive reinforcement pathways. In this pathway, substance use is thought to enhance positive affect. Recent research has focused on specific neurotic and disinhibitory personality dimensions that appear to be consistently linked with the initiation and maintenance of substance use; namely, hopelessness, anxiety sensitivity, impulsivity and sensation-seeking (Newton, Barrett, et al., 2016; Woicik, Stewart, Pihl, & Conrod, 2009). Recent research has shown that these personality traits were among the strongest predictors of adolescent binge-drinking (Whelan et al., 2014), with an Australian trial showing that a personality targeted intervention can have long-term effects in reducing alcohol and related harms (Newton, Conrod, et al., 2016).

### **Sociocultural factors**

Sociocultural factors play a widely varying role in substance use disorders. People's interest in and access to drugs are influenced by peers, the media and cultural norms about acceptable behaviour.

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Alcohol dependence is more prevalent in countries in which alcohol use is heavy.



At the broadest level, for example, we can look at cross-national variation in substance consumption. Some research suggests that there are commonalities in substance use across countries. For example, a cross-national study of alcohol and drug use among high school students in 36 countries found that alcohol was the most common substance used across countries, despite great variation in the proportions of students who consumed alcohol, ranging from 32 percent in Zimbabwe to 99 percent in Wales (Smart & Ogburne, 2000). In all but two of the countries studied, cannabis was the next most commonly used drug. In those countries where cannabis was used most often (with more than 15 percent of high school students having ever used cannabis), there were also higher rates of use of amphetamines, ecstasy and cocaine.

Despite the commonalities across countries, other research documents cross-national differences in alcohol consumption. For example, statistics from the World Health Organization indicate that Australia, along with Europe and Argentina, has one of the highest rates of per capita alcohol consumption. Much of South and North America consume moderate rates of alcohol, whereas most of Africa and Asia tend to consume alcohol at lower rates (World Health Organization, 2011). Cultural attitudes and patterns of drinking thus influence the likelihood of drinking heavily and therefore of abusing alcohol. One finding that seems quite similar across different cultures is that men consume more alcohol than women. An analysis conducted by the International Research Group on Gender and Alcohol found that men drank more than women in Australia, Canada, the Czech Republic, Estonia, Finland, Israel, the Netherlands, Russia, Sweden and the United States. Despite this consistency in gender differences, there was a large disparity across countries in the extent to which men drank more than women. For example, men drank three times more than women in Israel but only one and a half times more than women in the Netherlands (Wilsnack et al., 2000). These findings suggest that cultural

prescriptions about drinking by men and women are important to consider. However, as noted above, in more recent years the rates of consumption and alcohol-related problems have converged among men and women (Slade et al., 2016), with similar findings of convergence in cannabis use and harms (Chapman et al., Accepted).

Ready availability of the substance is also a factor. Findings from the Monitoring the Future Study, an ongoing study begun in the 1960s that surveys over 50 000 high school students in the United States every year, indicate that the greater reported availability of particular drugs or alcohol corresponded to greater use of those drugs (Johnston, O'Malley, Bachman, Schulenberg, & Miech, 2014). Meanwhile, Australian research also shows that increased alcohol outlet density and extended alcohol trading hours are correlated with increased alcohol use and alcohol-related harms (Chikritzhs, Catalano, Pascal, & Henrickson, 2007). This is one of the reasons that taxes on alcohol and cigarettes are increased so frequently. Increasing the price of alcohol and cigarettes has been robustly associated with decreases in their use internationally (Wagenaar, Salois, & Komro, 2009; Wakefield et al., 2014).

Family factors are important as well. One review focused on several different parental variables and their associations with reduced adolescent alcohol use. Several parental factors were associated with a delay in the initiation of adolescent alcohol use, as well as reduced levels of adolescent drinking, including: parental support, involvement, modelling and monitoring; limiting availability of alcohol to the child; parent-child relationship quality; general discipline; and general communication (Newton, Conrod, et al., 2016; Ryan, Jorm, & Lubman, 2010).

The social setting in which a person operates can also affect substance use. For example, studies of smokers in daily life show that they are more likely to smoke with other smokers than with non-smokers. In addition, smoking was more likely to occur in or outside bars and restaurants or at home, rather than in the workplace or in others' homes (Shiffman et al., 2002; Shiffman, Paty, Gwaltney, & Dang, 2004).

Other studies showed that having friends who smoke predicts smoking. In longitudinal studies, peer-group identification in Year 7 predicted smoking in Year 8 (Sussman et al., 1994) and increased drug use over a three-year period (Chassin, Curran, Hussong, & Colder, 1996). Peer influences are also important in promoting alcohol and cannabis use (Hussong et al., 2001; Stice, Barrera, & Chassin, 1998; Wills & Cleary, 1999).

These findings support the idea that social networks influence a person's drug or alcohol behaviour. However, other evidence indicates that people who are inclined to develop substance use disorders may actually select social networks that conform to their own drinking or drug use patterns. Thus, we have two broad explanations for how the social environment is related to substance use disorders: a social influence model and a social selection model. A longitudinal study of over 1200 adults designed to test which model best accounted for drinking behaviour found support for both models (Bullers, Cooper, & Russell, 2001). A person's social network predicted individual drinking, but individual drinking also predicted subsequent social network drinking. In fact, the social selection effects were stronger, indicating that people often choose social networks with drinking patterns similar to their own. No doubt the selected networks then support or reinforce their drinking.

Another variable to be considered is the media. The media also has an effect on alcohol and cigarette use. Research has consistently shown that banning or regulating alcohol and tobacco advertising results in a reduction in use. Tobacco advertising was banned in Australia in 1976. However, smoking on television and in movies has not been regulated, despite strong evidence to suggest that viewing smoking in movies promotes smoking initiation among adolescents (Dalton et al., 2003). Alcohol advertising is regulated in Australia, but there are concerns about the amount of alcohol advertising linked with sport in Australia. Recent data presented by the Royal Australasian College of Physicians, for example, indicate that each year 26.9 million children view AFL, NRL and cricket broadcasts, and during this time these children will be exposed to 50.9 million instances of alcohol advertising.

A billboard in Melbourne advertising sparkling wine



## 7.3 Treatment of substance use disorders

**LEARNING OUTCOME 7.3** Describe the approaches to treating substance use disorders, including psychological treatments, medications and drug substitution treatments.

In 2014–15, around 850 alcohol and other drug treatment services provided just over 170 000 treatment episodes to around 115 000 clients (AIHW, 2016a). This equates to approximately 1 in 200 people seeking treatment for alcohol and other drugs in the Australian population. Two-thirds of those seeking treatment were males and 15 percent were Indigenous. The main drugs of concern were alcohol (38 percent of treatment episodes) and cannabis (24 percent of treatment episodes), followed by amphetamines (20 percent of treatment episodes) and heroin (6 percent of treatment episodes). More than half, however, were concerned about more than one drug. While there was variability according to different substances, overall the main treatment delivery setting was a non-residential treatment facility, followed by residential treatment facilities and outreach treatment facilities. Types of alcohol and drug treatment services include counselling, non-residential, residential and hospital-based withdrawal services, residential and therapeutic day rehabilitation, and care and recovery coordination, as well as pharmacotherapy. There are also services available that are specific to youth and Indigenous populations. Most services in Australia adopt a harm-minimisation, rather than an abstinence-based, approach to treatment.

### Treatment of alcohol use disorder

Data from the 2007 NSMHWB indicate that one in five people who met criteria for an alcohol use disorder in the previous 12 months had sought treatment (Teesson et al., 2010) and this reduced to one in 10 among those aged 16–24 years (Mewton et al., 2011). Among those with a lifetime alcohol use disorder, the median delay to first treatment was 18 years (Chapman, Slade, Hunt, & Teesson, 2015).

We have a long way to go in developing and providing effective treatments for alcohol use disorders, and making sure that people who need treatment can get it. Of those who do get treatment, counselling, withdrawal management and assessment only are the most frequently accessed services.

### **Withdrawal management**

After a pattern of daily high alcohol use, withdrawal symptoms may occur when a person tries to stop drinking. These symptoms may be mild and tolerable or they may cause significant distress and require treatment. The onset of withdrawal symptoms from alcohol usually occurs from 6 to 12 hours after the last drink (Haber, Lintzeris, Proude, & Lopatko, 2009). Withdrawal symptoms can include nausea, vomiting, diarrhoea, sweating, fever, tremors, anxiety, psychomotor agitation and disturbed sleep. Severe withdrawal symptoms from alcohol can include dehydration, seizures, hallucinations and delirium. Often alcohol withdrawal syndromes are short-lived but, for some, can increase in severity over 48 to 72 hours after abstinence is instated. For heavy drinkers and those who have experienced previous alcohol withdrawal symptoms, close monitoring during the withdrawal period is recommended. Withdrawal services can be ambulatory (offered in a person's home environment or in supported accommodation), community residential-based or inpatient hospital-based. Supportive care during withdrawal should be provided, which includes the provision of information about alcohol withdrawal and symptoms, counselling to assist the patient in maintaining motivation and managing symptoms and monitoring of diet, nutrition and rehydration. Thiamine (vitamin B1) supplements should also be provided as a means of preventing Wernicke's encephalopathy, which is characterised by neurological symptoms due to the depletion of thiamine stores in some people with alcohol dependence. Benzodiazepines may also be recommended, as well as other medications to manage more severe symptoms (such as antipsychotic medications for hallucinations).

### **Cognitive and behavioural treatments**

Contingency management therapy is a cognitive-behavioural treatment for alcohol and drug use disorders that involves teaching people and those close to them to reinforce behaviours inconsistent with drinking — for example, taking the drug disulfiram (discussed later in the chapter) and avoiding situations associated with drinking in the past. It is based on the belief that environmental contingencies can play an important role in encouraging or discouraging drinking. Vouchers are provided for not using a substance (alcohol, cocaine, heroin, cannabis; verified by urine samples) and the tokens are exchangeable for things that the person would like to have more of (Dallery, Silverman, Chutuape, Bigelow, & Stitzer, 2001; Katz, Gruber, Chutuape, & Stitzer, 2001; Silverman et al., 1996). This therapy also includes teaching job-hunting and social skills, as well as assertiveness training for refusing drinks. For socially isolated people, assistance and encouragement are provided to establish contacts with other people who are not associated with drinking.

Relapse prevention is another cognitive-behavioural treatment that has been effective with alcohol and drug use disorders. It can be a standalone treatment or part of other interventions. Broadly, the goal is to help people avoid relapsing into drinking or drug use once they have stopped. Marlatt and Gordon (1985) developed an approach to treatment called relapse prevention specifically to prevent relapse in substance use disorders. In this approach, people dependent on alcohol are encouraged to believe that a lapse will not inevitably precipitate a total relapse and should be regarded as a learning experience rather than as a sign that the battle is lost, a marked contrast from the Alcoholics Anonymous perspective. This non-catastrophising approach to relapse after therapy — falling off the wagon — is important because the overwhelming majority of people who are dependent on alcohol and become abstinent experience one or more relapses over a four-year period (Polich, Armor, & Braiker, 1980). People dependent on alcohol examine sources of stress in their work, family and relationships so that they can become active and responsible in anticipating and resisting situations that might lead them into excessive drinking (Marlatt, 1983; Sobell, Toneatto, & Sobell, 1990). The sources of stress that precipitate a relapse in alcohol use disorder may be different for men and women. For women, marital stress is a predictor of relapse. For men, however, marriage seems to protect them from relapse (Walitzer & Dearing, 2006).

There are also effective online interventions for alcohol use disorders with co-occurring anxiety and depression. The evidence base in this area is growing as the internet provides a less stigmatised way to deliver treatment. Many of the programs are based on cognitive-behavioural principles and have shown positive outcomes in adults and young adults (Champion, Newton, & Teesson, 2016; Deady, Mills, Teesson, & Kay-Lambkin, 2016).

### **Couples therapy**

Behaviourally oriented marital or couples therapy (O'Farrell & Fals-Stewart, 2000) has been found to achieve some reductions in problem drinking, even a year after treatment has stopped, as well as some improvement in couples' distress generally (McCrary & Epstein, 1995). This appears to be effective for straight, gay and lesbian couples (Fals-Stewart, O'Farrell, & Lam, 2009). This treatment combines the skills covered in individual cognitive-behavioural therapy, with a focus on the couple's relationship and dealing with alcohol-related stressors together as a couple. A meta-analysis of 12 studies found that behaviourally oriented couples therapy was more effective than individual treatment approaches (Powers, Vedel, & Emmelkamp, 2008).

### **Motivational interviewing**

Specifically for problematic alcohol use, motivational interviewing consists of addressing any ambivalence towards changing drinking habits or other behaviours. During motivational interviewing, the therapist facilitates changes in behaviour using non-confrontational approaches. Motivational interviewing can be delivered as a standalone treatment or as an adjunct to other psychosocial or pharmacological treatments. A recent meta-analysis indicated that among young drinkers, motivational interviewing is only minimally effective (Foxcroft, Coombes, Wood, Allen, & Almeida Santimano, 2014).

### **Moderation in drinking**

The popular belief has been that people with alcohol use disorder have to abstain completely if they are to be successfully treated, for they presumably have no control over drinking once they take that first drink. Considering the social difficulty of avoiding alcohol altogether, it may be preferable to teach a person who does not use alcohol in an extreme fashion to drink with moderation. The term **controlled drinking** was introduced into the domain of alcohol treatment by Mark and Linda Sobell, who developed the *guided self-change* approach to treatment (Sobell & Sobell, 1993). The basic assumption is that people have more potential control over their immoderate drinking than they typically believe and that heightened awareness of the costs of drinking to excess as well as of the benefits of abstaining or cutting down can help. For example, getting the person to delay 20 minutes before taking a second or third drink can help him or her reflect on the costs versus the benefits of drinking to excess. Evidence supports the effectiveness of this approach in helping people moderate their intake and otherwise improve their lives (Sobell & Sobell, 1993). A randomised controlled clinical trial demonstrated that guided self-change was just as effective as an individual or group treatment (Sobell, Sobell, & Agrawal, 2009).

### **Alcoholics Anonymous**

The largest and most widely known self-help group in the world is Alcoholics Anonymous (AA), founded in 1935 by two recovering alcoholics. It has 1900 chapters in Australia, servicing about 18 000 members, making AA the most widely available program for alcohol dependent people in Australia (Haber et al., 2009).

Each AA chapter runs regular and frequent meetings at which newcomers rise to announce that they are alcoholics and give testimonials, relating the stories of their problems with alcohol and indicating how their lives are better now. The group provides emotional support, understanding and close counselling as well as a social network. Members are urged to call on one another around the clock when they need companionship and encouragement not to relapse. Programs modelled after AA are available for

other substances, for example, Narcotics Anonymous, Cocaine Anonymous and Cannabis Anonymous.

The AA program tries to instil in each member the belief that alcohol dependence is a disease that can never be cured and that continuing vigilance is necessary to resist taking even a single drink, lest uncontrollable drinking begin all over again. Even if the person has not consumed any alcohol for 15 years or more, the designation 'alcoholic' is still necessary according to the tenets of AA, since the person is always an alcoholic, always carrying the disease, even if it is currently under control.

The spiritual aspect of AA is apparent in the 12 steps of AA, and there is evidence that belief in this philosophy is linked with achieving abstinence (Fiorentine & Hillhouse, 2000; Tonigan, Miller, & Connors, 2000), although some studies have shown that this is not necessary (Haber et al., 2009). A review of eight randomised controlled clinical trials found little benefit of AA over other types of treatment, including motivational enhancement, inpatient treatment, couples therapy or cognitive-behavioural therapy (Ferri, Amato, & Davoli, 2008). In addition, AA has high dropout rates, with new members encouraged to attend 90 meetings in 90 consecutive days.

Alcoholics Anonymous is the largest self-help group in the world. At their regular meetings, newcomers rise to announce their addiction and receive advice and support from others.



## SMART Recovery

Self Management and Recovery Training (SMART) is a not-for-profit self-help group aimed at facilitating recovery from any addictive behaviour (Haber et al., 2009). SMART Recovery is a non-spiritual alternative to AA, with over 50 groups currently operating in Australia. It is built on cognitive-behavioural principles and uses a four-point recovery program that is designed to enhance motivation and teach techniques that help manage lifestyle and behavioural difficulties. The four points include:

1. enhancing and maintaining motivation to abstain
2. coping with urges
3. problem solving (managing thoughts, feelings and behaviours)
4. lifestyle balance (balancing momentary and enduring satisfaction).

## Medications

Three pharmacotherapies — naltrexone, acamprosate and disulfiram — are available in Australia for the treatment of alcohol dependence. Naltrexone is an opioid receptor antagonist which blocks the activity of endorphins that are stimulated by alcohol, thus reducing the craving for it. Naltrexone has been shown to reduce the rates of relapse and increase periods of abstinence in people with alcohol dependence (Haber et al., 2009). Evidence is mixed regarding whether this drug is more effective than a placebo in reducing

drinking when it is the only treatment (Krystal, Cramer, Krol et al., 2001). But it does appear to add to overall treatment effectiveness when combined with cognitive-behavioural therapy (Pettinati, Oslin, Kampman et al., 2010; Streeton & Whelan, 2001; Volpicelli, Rhines, Rhines et al., 1997; Volpicelli, Watson, King, Shermen, & O'Brien, 1995).

Another drug shown to be effective is acamprosate. A review of data from all published double-blind, placebo-controlled clinical trials of acamprosate for people dependent on alcohol suggests that it is highly effective (Mason, 2001). A meta-analysis comparing the effectiveness of acamprosate and naltrexone found them to be equally effective (Kranzler & Van Kirk, 2001). Although its action is not completely understood, researchers believe that it impacts the glutamate and GABA neurotransmitter systems and thereby reduces the cravings associated with withdrawal. Acamprosate takes one to two weeks to take effect. Acamprosate and naltrexone can be administered simultaneously, with evidence suggesting that the combination may be better than acamprosate alone, but not more effective than naltrexone alone (Haber et al., 2009).

Some people who are in treatment for alcohol use disorder, inpatient or outpatient, take disulfiram, a drug that discourages drinking by causing vomiting, dizziness, irregular heartbeat, breathlessness and headache if alcohol is ingested. The evidence for the effectiveness of disulfiram is weaker than that for naltrexone and acamprosate (Haber et al., 2009). A relatively new drug, topiramate, also appears to be promising in treating alcohol dependence (Johnson et al., 2008; Johnson et al., 2007).

## Treatments for smoking

The numerous laws that currently prohibit smoking in almost all public places are part of a social context that provides incentives and support to stop smoking. In addition, people are more likely to quit smoking if other people around them quit. A longitudinal study of over 12 000 people demonstrated that if people in one's social network quit smoking (spouses, siblings, friends, co-workers), the odds that a person will quit smoking are much greater (Christakis & Fowler, 2008). For example, if a person's spouse stopped smoking, his or her chances of continued smoking decreased by nearly 70 percent. In short, peer pressure to quit smoking appears to be as effective as peer pressure to start smoking once was.

Some smokers who want to quit attend smoking clinics or consult with professionals for other specialised smoking-reduction programs. Even so, it is estimated that only about half of those who go through smoking-cessation programs succeed in abstaining by the time the program is over; only a very small percentage of those who have succeeded in the short term actually remain non-smoking after a year (Brandon, Vidrine, & Litvin, 2007).

## Psychological treatments

Probably the most widespread psychological treatment consists of a physician telling the person to stop smoking. Each year millions of smokers are given this counsel — because of hypertension, heart disease, lung disease or diabetes, or on general grounds of preserving or improving health. There is some evidence that a physician's advice can get some people to stop smoking, at least for a while, especially when the person also chews nicotine gum (Law & Tang, 1995). Motivational interviewing has been tried to help people quit smoking, but results from a meta-analysis indicate that it is only modestly effective (Hettema & Hendricks, 2010; Lai, Cahill, Qin, & Tang, 2010).

One treatment approach that seems to work is called scheduled smoking (Compas, Haaga, Keefe, Leitenberg, & Williams, 1998). The strategy behind this approach is to reduce nicotine intake gradually over a period of a few weeks by getting smokers to agree to increase the time between cigarettes. For example, during the first week of treatment, a one-pack-a-day smoker would be put on a schedule allowing only 10 cigarettes per day; during the second week, only five cigarettes a day would be allowed; and during the third week, the person would taper off to zero. The cigarettes would have to be smoked on a schedule provided by the treatment team, not when the smoker feels an intense craving. In this way, the person's smoking behaviour is controlled by the passage of time rather than by urges, mood states or

situations. Smokers who are able to stay with the agreed-upon schedule showed a 44 percent abstinence rate after one year (Cinciripini et al., 1994).

### Nicotine replacement treatments and medications

Reducing a smoker's craving for nicotine by providing it in a different way is the goal of nicotine replacement treatments (NRT). Attention to nicotine dependence is clearly important because the more cigarettes people smoke daily, the less successful they are at quitting. Nicotine may be supplied in gum, patches, inhalers or e-cigarettes. The idea is to help smokers endure the nicotine withdrawal that accompanies any effort to stop smoking. Although nicotine replacement alleviates withdrawal symptoms — which justifies its use in gum and in the nicotine patches to be described next — the severity of withdrawal is only minimally related to success in stopping smoking (Ferguson, Shiffman, & Gwaltney, 2006; Hughes, Higgins, & Hatsukami, 1990).

Nicotine gum, available over the counter, is absorbed much more slowly and steadily than that in tobacco. The long-term goal is for the former smoker to be able to cut back on the use of the gum as well, eventually eliminating reliance on nicotine altogether. However, in doses that deliver an amount of nicotine equivalent to smoking one cigarette an hour, the gum causes cardiovascular changes, such as increased blood pressure, that can be dangerous to people with cardiovascular diseases. Nevertheless, some experts believe that even prolonged, continued use of the gum is healthier than obtaining nicotine by smoking, because the carcinogens are avoided (de Wit & Zacny, 2000).

Nicotine patches, also available over the counter, contain a polyethylene patch taped to the arm that slowly and steadily releases the drug into the bloodstream transdermally (through the skin) and then to the brain. An advantage of the patch over nicotine gum is that the person need only apply one patch each day and not remove it until applying the next patch, making compliance easier. Treatment can be effective after eight weeks of use for most smokers (Stead, Perera, Bullen, Mant, & Lancaster, 2008), with smaller and smaller patches used as treatment progresses. A drawback is that people who continue smoking while wearing the patch risk increasing the amount of nicotine in their body to dangerous levels.

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Nicotine patches are available over the counter to help relieve withdrawal symptoms.



Evidence suggests that the nicotine patch is superior to the placebo patch in terms of both abstinence and subjective craving (Hughes et al., 1990). A meta-analysis of 111 trials of all types of nicotine replacement treatments (patch, gum, nasal spray, inhaler, tablets) found that NRT was more effective than placebo in smoking cessation (Stead et al., 2008). People who begin to stop smoking after wearing the patch but before dedicated cessation efforts begin are more likely to remain abstinent from smoking at the end of NRT (Rose, Herskovic, Behm, & Westman, 2009). However, NRT is not a panacea. Abstinence rates are only about 50 percent at 12-month follow-ups. The manufacturers state that the patch is to be used only as part of a psychological smoking-cessation program and then for not more than three months at a time. In addition, these types of nicotine replacement treatments are not effective with adolescents (Curry, Mermelstein, & Sporer, 2009).

NRT plus medication or psychological treatment appears to be more effective than NRT alone (Hughes, 2009; Rose & Behm, 2014). For example, combining the antidepressant medication bupropion and nicotine patches yielded a 12-month abstinence rate of 35 percent in one study (Jorenby, Leischow, Nides et al., 1999) but less promising results in others (Hughes, Stead, & Lancaster, 2004; Killen et al., 2006). Another drug, varenicline, may be effective in combination with behavioural treatment and even more effective than bupropion (Cahill, Stead, & Lancaster, 2007; Tonstad et al., 2006).

There is a current debate about whether e-cigarettes are an effective form of NRT. Results from one longitudinal study found that only 10 percent of people using e-cigarettes had quit smoking one year later (Grana, Popova, & Ling, 2014). An internet survey of over 400 people who used e-cigarettes found that 22 percent of people had stopped smoking after one month and 46 percent had stopped at the one-year follow-up (Etter & Bullen, 2014). A randomised controlled clinical trial assigned people who wanted to quit smoking to either e-cigarettes, the nicotine patch or placebo e-cigarettes. The numbers of people who quit smoking were very small in all three groups, so small in fact that the researchers did not have the statistical power to detect any significant differences between the groups (Bullen et al., 2013). Clearly, more research is needed, but thus far, e-cigarettes do not appear to be more effective than other forms of NRT.

## Treatment of drug use disorders

Central to the treatment of people who use drugs such as heroin and cocaine is withdrawal from the drug itself. Heroin withdrawal reactions range from relatively mild bouts of anxiety, nausea and restlessness for several days to more severe and frightening bouts of delirium and panic. The type of reaction depends primarily on the purity of the heroin that the person has been using. The cravings for the substance often remain even after the substance has been removed.

### Psychological treatments

In the first direct comparison in a controlled study, the antidepressant medication desipramine and cognitive-behavioural therapy (CBT) were both found to be somewhat effective in reducing cocaine use as well as in improving a person's family, social and general psychological functioning (Carroll, Rounsaville, et al., 1994; Carroll, Rounsaville, Nich, Gordon, & Gawin, 1995). In this 12-week study, desipramine was better than a placebo for people with a low degree of dependence on cocaine, whereas CBT was better for people with a high degree of dependence.

In this study, people receiving CBT learned how to avoid high-risk situations (e.g., being around people using cocaine), recognise the lure of the drug for them and develop alternatives to using cocaine (e.g., recreational activities with non-users). People also learned strategies for coping with the craving and for resisting the tendency to regard a slip as a catastrophe (relapse prevention training). A more recent study of computer-based CBT compared to standard substance abuse counselling found that CBT was more effective in helping people remain abstinent from cocaine (Carroll et al., 2014) for up to six months after treatment. Despite this promise, we are still far short of developing more effective and longer lasting treatments.

Contingency management with vouchers has shown promise for cocaine, heroin and cannabis use disorders (Dallery et al., 2001; Katz et al., 2001; Petry, Alessi, Marx, Austin, & Tardif, 2005; Silverman et al., 1996). For example, a randomised treatment trial for people with cannabis use disorder compared a voucher treatment, CBT and CBT plus vouchers (Budney, Moore, Rocha, & Higgins, 2006). During the treatment, people who received the voucher treatment were more likely to remain abstinent than those in the CBT treatment or in the CBT plus vouchers treatment. After treatment was over, however, people who received CBT plus vouchers were most likely to remain abstinent. Thus, vouchers appear to work in the short term, but CBT appears to be an effective component of treatment for cannabis use disorder in the long term with respect to maintaining abstinence after treatment is over.

Studies of contingency management for cocaine use disorder find that it is associated not only with a greater likelihood of abstinence but also with a better quality of life (Petry, Alessi, & Hanson, 2007). In an analysis that looked at four different studies of contingency management treatment for cocaine use disorder, the researchers found that people who received contingency management treatment were more likely to remain abstinent than people who received treatment as usual and that the duration of their abstinence during treatment was related to a higher quality of life after treatment. A meta-analysis of four randomised controlled clinical trials comparing contingency management, day treatment or both treatments (combined condition) for cocaine use among homeless people found that the combined treatment and contingency management were both more effective than day treatment alone (Schumacher, Milby, Wallace et al., 2007).

Motivational enhancement therapy has also shown promise. This treatment involves a combination of CBT techniques and techniques associated with helping clients generate solutions that work for themselves. A meta-analysis of this treatment found that it was effective for both alcohol and drug use disorders (Burke, Arkowitz, & Menchola, 2003). Another study found that motivational enhancement combined with CBT and contingency management was an effective treatment package for young people (ages 18–25) who were dependent on cannabis (Carroll et al., 2006).

Self-help residential homes are another psychological approach to treating heroin and other types of drug use disorders. Drug-rehabilitation homes share the following features:

- separation of people from previous social contacts, on the assumption that these relationships have been instrumental in maintaining the drug use disorder
- a comprehensive environment in which drugs are not available and continuing support is offered to ease the transition from regular drug use to a drug-free existence
- the presence of charismatic role models, people formerly dependent on drugs who appear to be meeting life's challenges without drugs
- direct, often intense, confrontation in group therapy, in which people are goaded into accepting responsibility for their problems and for their drug habits and are urged to take charge of their lives
- a setting in which people are respected as human beings rather than stigmatised as failures or criminals.

There are several obstacles to evaluating the efficacy of residential drug-treatment programs. Because the dropout rate is high, those who remain cannot be regarded as representative of the population of people addicted to illegal drugs; their motivation to stop using drugs is probably much stronger than that of people who don't volunteer for treatment or people who drop out. Any improvement participants in these programs make may reflect their strong motivation to quit more than the specific qualities of the treatment program. Such self-regulating residential communities do, however, appear to help a large number of those who remain in them for a year or so (Institute of Medicine, 1990; Jaffe, 1985).

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Group therapy in residential settings is frequently used to treat heroin addiction.



### Drug replacement treatments and medications

Two widely used programs for heroin use disorder involve the administration of *heroin substitutes*, drugs chemically similar to heroin that can replace the body's craving for it, or *opiate antagonists*, drugs that prevent the user from experiencing the heroin high. An antagonist is a drug that dampens the activity of neurotransmitters and an agonist is a drug that stimulates neurotransmitters. The first category includes **methadone**, levomethadyl acetate and buprenorphine, synthetic narcotics designed to take the place of heroin. Since these drugs are themselves addicting, successful treatment essentially converts the person's dependence on heroin into dependence on a different substance. This conversion occurs because these synthetic narcotics are **cross-dependent** with heroin; that is, by acting on the same central nervous system receptors, they become a substitute for the original dependency. Abrupt discontinuation of methadone results in its own pattern of withdrawal reactions, but because these reactions are less severe than those of heroin, methadone has potential for weaning heroin users altogether from drug dependence (Strain, Bigelow, Liebson, & Stitzer, 1999).

Treatment with the opiate antagonists involves naltrexone, discussed above. First, people are gradually weaned from heroin. Then they receive increasing dosages of naltrexone, which prevents them from experiencing any high should they later take heroin. This drug works because it has great affinity for the receptors to which opiates usually bind; their molecules occupy the receptors without stimulating them. This leaves heroin molecules with no place to go and so heroin does not have its usual effect on the user. As with methadone, however, treatment with naltrexone involves frequent (daily) and regular visits to a clinic, which requires motivation. In addition, people may not lose the craving for heroin for some time. Both clinical effectiveness and treatment compliance can be increased by adding a contingency management component to the therapy (Carroll, Ball, Nich et al., 2001). Giving people vouchers that they can exchange for food and clothing in return for taking naltrexone and having drug-free urine samples markedly improves effectiveness. Treatment with a heroin substitute usually involves going to a drug-treatment clinic and swallowing the drug in the presence of a staff member, once a day for

methadone and three times a week for levomethadyl acetate and buprenorphine. There is some evidence that methadone maintenance can be carried out more simply and just as effectively by weekly visits to a physician (Fiellin et al., 2001). The effectiveness of methadone treatment is improved if a high (80- to 100-milligram) dose is used as opposed to the more typical 40- to 50-milligram dose (Strain et al., 1999) and if it is combined with regular psychological counselling (Ball & Ross, 1991). Drug treatment experts generally believe that treatment with heroin substitutes is best conducted in the context of a supportive social interaction, not merely as a medical encounter (Lilly, Quirk, Rhodes, & Stimson, 2000).

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Methadone is a synthetic heroin substitute. People formerly diagnosed with heroin use disorder come to clinics each day and swallow their dose.



Since methadone does not provide a euphoric high, many people will return to heroin if it becomes available to them. In an effort to improve outcomes, researchers have tried adding contingency management to the usual treatment at methadone clinics. In one randomised controlled trial (Pierce, Petry, Stitzer et al., 2006), people receiving methadone from a clinic could draw for prizes each time they submitted a (carefully supervised and obtained) urine sample that had no trace of illegal drugs or alcohol. Prizes ranged from praise to televisions. People who were in the contingency management group were more likely to remain drug-free than those people who received only usual care from the methadone clinic. Of course, it remains to be seen whether such abstinence gains can be maintained after treatment ends and therapists are no longer providing such incentives.

Unfortunately, many people drop out of methadone programs, in part because of side effects such as insomnia, constipation, excessive sweating and diminished sexual functioning. The stigma associated with going to methadone clinics is also linked to dropout rates, as illustrated in the clinical case of James described earlier.

Given the limitations of heroin substitutes such as methadone and other opiate antagonists, researchers have been searching for alternative medications. Buprenorphine (Suboxone) is a medication that actually contains two agents: buprenorphine and naloxone. Buprenorphine is a partial opiate agonist, which

means it does not have the same powerfully addicting properties as heroin, which is a full agonist. Naloxone is an opiate antagonist, often used in emergency rooms for opiate or heroin overdoses. This unique combination in Suboxone does not produce an intense high, is only mildly addictive and lasts for as long as three days. Heroin users do not need to go to a clinic to receive this medication since it can be prescribed to individuals. Thus, this treatment avoids the stigma associated with visiting methadone clinics. Suboxone is effective at relieving withdrawal symptoms and because it lasts longer than methadone, researchers are hopeful that relapse will be less likely. Still, some users may miss the more euphoric high associated with heroin, thus hastening a relapse.

Drug replacement does not appear to be an effective treatment for cocaine use disorder. A meta-analysis of nine randomised controlled clinical trials of stimulant medication as a treatment for cocaine revealed little evidence that this type of medication is effective (Castells et al., 2007).

Researchers were hopeful that a vaccine to prevent the high associated with cocaine use would be effective. The vaccine contains tiny amounts of cocaine attached to otherwise harmless pathogens. The body's immune system responds to this invasion by developing antibodies that then squelch the cocaine. It was hoped that with repeated exposure, the antibodies would be able to keep a good deal of the cocaine from reaching the brain. However, a randomised controlled clinical trial with over 100 people addicted to cocaine was not particularly promising (Martell et al., 2009). First, for the vaccine to be effective, people needed to receive five shots and only half of the sample followed through. Second, just over a third of people receiving all shots developed enough antibodies to keep cocaine from reaching the brain. And finally, although about half of the sample used cocaine less, the vaccine did not help stave off cravings for cocaine. Clearly, additional work needs to be done in this area.

### **Treatments for methamphetamine use**

Developing effective treatments for methamphetamine dependence remains a challenge for the field. People like Anton, described in the clinical case earlier, do not have many places to turn for treatment. The largest effort to date is a randomised controlled clinical trial conducted across eight different sites referred to as the Methamphetamine Treatment Project (Rawson, Martinelli-Casey, Anglin et al., 2004). This study compared a multifaceted treatment called Matrix with treatment as usual. The Matrix treatment consisted of 16 CBT group sessions, 12 family education sessions, four individual therapy sessions and four social support group sessions. Treatment as usual (TAU) consisted of the best available treatment currently offered at the eight outpatient clinics. This varied quite a bit across the sites, with some offering individual counselling and others offering group counselling; some offering four weeks of treatment and others offering 16 weeks. Results of the study are somewhat supportive of the Matrix treatment. Compared to those in TAU, those people receiving Matrix stayed in treatment longer and were less likely to use methamphetamine during treatment (confirmed with urine analysis). Unfortunately, at the end of treatment and at the six-month follow-up, people who received Matrix were no less likely to have used methamphetamine than those in TAU. The good news is that all participants were less likely to use methamphetamine after six months, regardless of whether they received Matrix or TAU. Although these results are promising, additional work is clearly needed to develop effective treatments for methamphetamine problems.

In an Australian study comparing treatment outcomes for methamphetamine users, participants attended either community-based detoxification or residential rehabilitation and were compared with a quasi-control group of methamphetamine users from the community (McKetin et al., 2012). When compared with the quasi-control group, detoxification did not have any effect on methamphetamine use at three months, one year or three years follow-up. Residential rehabilitation, on the other hand, produced large reductions in methamphetamine use at three months follow-up, which were also evident one year following treatment. These effects, however, were no longer evident at three years follow-up. The authors concluded that residential rehabilitation results in a time-limited decrease in methamphetamine use, whereas the detoxification alone did not appear effective.

## 7.4 Prevention of substance use disorders

**LEARNING OUTCOME 7.4** Delineate the major approaches to preventing substance use disorders.

Prevention of substance use disorders can be universal, selective or indicated in focus. Universal prevention focuses on whole populations, regardless of risk for the development of substance use and related problems. Selective prevention strategies target subgroups of populations that are determined to be at risk for developing substance use and related problems. Such risk factors may include personality styles or sociocultural characteristics. Finally, indicated prevention strategies are delivered to individuals already experiencing early signs and symptoms of problematic substance use. Though experimenting with alcohol and drugs, and perhaps demonstrating some symptoms of a substance use disorder, individuals identified at this stage would not have passed the threshold for a clinical diagnosis of a substance use disorder.

Due to the inclusion of all members of a given group, regardless of relative risk, the effect sizes for universal prevention strategies are often small. Small effect sizes in the context of delaying the initiation of substances, however, have been associated with significant savings in terms of social costs (Caulkins, Pacula, Paddock, & Chiesa, 2004) and so the real world impact is significant. Australian research, for example, has indicated that universal preventive interventions are consistently more cost-effective than treatment when focusing on harms related to alcohol (Cobiac, Vos, Doran, & Wallace, 2009). Similarly, research based in the US suggests that every dollar spent on universal substance abuse prevention results in savings ranging from \$2.00 to \$19.64 (Swisher, Scherer, & Yin, 2004). Selective prevention strategies, on the other hand, target subsets of a population that have been identified as being at an increased risk of developing substance use problems. Although selective prevention programs have the potential to address risk for substance use problems early on, screening for at-risk individuals can be costly in terms of time and money and may entail labelling and stigmatisation issues (Offord & Kraemer, 2000). The following section focuses on universal prevention only.

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Examples of the health warnings now required for cigarette packages since Australian regulations were introduced in Australia in 2012



### Online prevention for substance use disorders

In recent years, technology-based preventions and interventions facilitated by computers or the internet have emerged as promising strategies. This is due to the many advantages technology affords over traditional methods, including increased accessibility, affordability and greater feasibility of use (Champion, Newton, Stapinski, & Teesson, 2016). Two systematic reviews composed of mainly school-based interventions indicated that school-based programs facilitated by computers or the internet can have preventive effects on alcohol use, but the findings with regards to illicit drug use were mixed (Champion, Newton, Barrett, & Teesson, 2013; Champion et al., 2016). One other review focusing on very brief internet-delivered alcohol interventions in college settings indicated these programs appeared effective in reducing alcohol use (Leeman, Perez, Nogueira, & DeMartini, 2015). Embedding drug and alcohol interventions within serious educational games delivered via computers or the internet represents another novel approach to prevention that aims to maximise participant engagement and looks promising in terms of effectiveness.

There is now also meta-analytic evidence for the effectiveness of computer and internet interventions for smoking (Rooke, Thorsteinsson, Karpin, Copeland, & Allsop, 2010), alcohol use (Khadjesari, Murray, Hewitt, Hartley, & Godfrey, 2011) and illicit drug use (Tait, Spijkerman, & Riper, 2013). While these generally show positive effects, there are limitations to these approaches, especially in terms of adherence and the scientific quality of some of the studies (Kiluk et al., 2011). Overall, computer- and internet-facilitated programs for the prevention and intervention of substance use show increasing promise and would benefit from further research.

#### QUESTIONS

1. Is there evidence for the effectiveness of computer and internet prevention interventions for smoking, alcohol use and illicit drug use?
2. Is it possible that technology-based preventions and interventions facilitated by computers or the internet have emerged as promising strategies?

### School-based prevention

The content of school-based prevention programs for alcohol and drugs is typically based on social competence models or social norms approaches, and tends to include aspects of psychoeducation (Faggiano, Minozzi, Versino, & Buscemi, 2013). Social competence approaches tend to teach skills based on the principles of cognitive-behavioural therapy and might include social skills training, goal setting, problem solving and decision making. Social competence approaches to prevention may also teach cognitive skills to resist peer and media influences, enhance self-esteem, increase assertiveness, and cope with stress and anxiety. Social norms approaches correct over-estimations of the substance use of peers and society at large, and teach techniques for recognising high-risk situations, negative influences of peers, family and media, as well as drug refusal skills. Providing psychoeducation (i.e., information about alcohol and drugs) in isolation is not an effective prevention strategy, but can be used effectively in combination with either social competence or social norms approaches.

A recent review of the literature identified school-based alcohol and drug programs that are effective in an Australian context (Teesson, Newton, & Barrett, 2012). Four Australian programs were identified that had beneficial effects on both alcohol use and binge drinking: the School Health and Alcohol Harm Reduction Project (SHAHRP), CLIMATE Alcohol, CLIMATE Alcohol and Cannabis and the Gatehouse Project. Two programs had beneficial effects on cannabis use outcomes: CLIMATE Alcohol and Cannabis and the Gatehouse Project. Two of the identified programs had no beneficial effects on the substance use outcomes measured: Life Education and the Resilient Families Intervention. Several other international school-based universal prevention programs have also been shown to be effective

in reducing alcohol and drug use, including Project Alert (USA), All Stars (USA), Life Skills Training (USA), Skills for Adolescence (USA), Unplugged (Europe) and the Good Behaviour Game (USA and Europe).

Smoking interventions are widely implemented in school settings without rigorous evaluation (Stockings et al., 2016). Of the evaluation studies that exist, it appears that social influences and social competence approaches to preventing smoking can affect tobacco use in the short term, with some evidence that specific programs (such as Life Skills Training) can be effective over the long term (Thomas, McLellan, & Perera, 2015).

### **Prevention involving parents and families**

There is good evidence to suggest that family-based universal primary prevention programs are effective in delaying and reducing alcohol and tobacco use and emerging evidence to suggest that these programs are also effective in reducing illicit drug use. Family-based preventive interventions that include a psychosocial aspect designed to have an impact on a range of behaviours are more effective in reducing substance use and harms than those that focus exclusively on substance use behaviours (Foxcroft & Tsertsvadze, 2011; Petrie, Bunn, & Byrne, 2007). Active participation of the parents also appears fundamental to the success of the intervention (Petrie et al., 2007). There is also evidence to suggest that family-based interventions for substance use have the greatest impact when they involve children in early adolescence, after which the importance of the family diminishes and is replaced with more of a focus on peers. Finally, the evidence suggests that gender-specific interventions — for example, those targeting mother and daughter dyads — can also be effective in the short and medium term.

### **Mass media campaigns to prevent substance use**

There is little evidence to suggest that mass media campaigns have an effect on alcohol and illicit drug use, with some evidence to suggest that such campaigns may increase illicit drug use in young people (Stockings et al., 2016). There is moderate to strong evidence for the benefit of mass media campaigns to change other health behaviours, including smoking, physical activity, safe sex practices and road safety behaviours (Wakefield, Loken, & Hornik, 2010). However, these have not been replicated in the literature focusing on behaviours related to alcohol and drug use. It may be that different approaches informed by successful campaigns aimed at other health behaviours may be successfully applied to the prevention of alcohol and drug use behaviours in the future.

### **Policy approaches to prevent alcohol and tobacco use**

There is strong, convincing and consistent evidence to suggest that there is an inverse relationship between alcohol taxation and alcohol consumption (Elder et al., 2010; Wagenaar et al., 2009) and harms (Wagenaar, Tobler, & Komro, 2010). Alcohol outlet density is also positively associated with alcohol use and alcohol-related harms (Bryden, Roberts, McKee, & Petticrew, 2012; Popova, Giesbrecht, Bekmuradov, & Patra, 2009). There is also reasonably consistent evidence to suggest that *increasing* trading hours or days leads to increases in alcohol consumption and related harms, especially if the increase is greater than two hours (Hahn et al., 2010).

A recent review of 77 studies in 21 countries indicated that legislative smoking bans had a consistent positive impact on cardiovascular health outcomes and mortality associated with smoking-related illnesses on a national level (Frazer et al., 2016). This review found inconsistent evidence regarding the impact of legislative smoking bans on respiratory and perinatal health outcomes. In terms of smoking prevalence and tobacco consumption, the evidence was also inconsistent, with some studies not finding any additional changes in existing prevalence trends associated with legislative smoking bans.

A recent large study of tobacco control policies and smoking prevalence in Australia investigated the extent to which tobacco taxes, smoke-free laws, mass media campaigns, the advertising of nicotine replacement therapy and graphic warning labels affected smoking prevalence in Australia from 2001–11. Overall, the smoking prevalence for the study period reduced from 23.6 percent (in January 2001) to

17.3 percent (in June 2011). The strongest predictors of this decline in smoking were smoke-free laws, tobacco price increases and televised mass media campaigns, together explaining over three-quarters of the reduction in smoking (Wakefield et al., 2014). Other research has also found that graphic warning labels are effective in preventing smoking and helping people to quit, largely because they make clear the health consequences of smoking (Hammond, 2011; Huang, Chaloupka, & Fong, 2014). Plain packaging of tobacco products was introduced in Australia in December 2012. While it is still too early to make any strong claims about the impact of plain packaging on smoking prevalence in Australia, research indicates many beneficial outcomes in terms of reductions in public display of packs, calls to telephone support services aimed at smoking cessation and reported propensity to smoke (Daube, Eastwood, Mishima, & Peters, 2015). Meanwhile, Australian data indicates that smoking reduced 3.4 percent from 2012 to 2013, with a further 7.7 percent reduction from 2013 to 2014.

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## SUMMARY

### **7.1 Describe the epidemiology and symptoms associated with substance use disorders.**

Alcohol and drug use is common in Australia. The DSM-5 lists substance use disorders for alcohol and many other substances, with severity determined by the number of symptoms present. Alcohol use is particularly high among young people; men are historically more likely to drink alcohol than women, although this gender gap is no longer as evident in more recent cohorts. Alcohol is the leading risk factor contributing to burden of disease among young people.

Smoking remains widespread, although it has nearly halved in the past 20 years. Cigarette smoking causes a number of illnesses, including several cancers, heart disease and other lung diseases. Secondhand smoke (or passive smoke) is also linked to a number of serious health problems. Females are less likely to smoke than males and the rates of smoking have declined sharply among young people. E-cigarettes are rapidly becoming popular, particularly among young people. The health effects of these products are not yet known, but studies are underway.

Cannabis makes people feel relaxed and sociable, but it can also interfere with cognitive functioning. In addition, it has been linked to lung-related problems and impaired driving. It remains the most prevalently used illicit drug, particularly among younger people. Cannabis also has therapeutic benefits, including for those suffering from the side effects of chemotherapy and for those with AIDS, glaucoma, seizures, chronic pain and muscle spasms.

Opiates include heroin and other pain medications. Abuse of prescription pain medications has risen dramatically. Initial effects of opiates include euphoria; later, users experience a comedown. Death by overdose from opiates is a severe problem. Other problems include exposure to HIV and other infectious agents through the use of shared needles.

Amphetamines are stimulants that produce wakefulness, alertness and euphoria. Tolerance develops quickly. Methamphetamine is a synthesised amphetamine and use of the crystal form of methamphetamine has increased over recent years. Methamphetamine can damage the brain, including the hippocampus. Cocaine can increase sexual desire, feelings of wellbeing and alertness, but chronic use is associated with problems in relationships, paranoia and trouble sleeping, among other things.

LSD was a popular hallucinogen in the 1960s and 1970s, often billed as a mind-expanding drug. The mind-expanding drug of the 1990s became ecstasy.

### **7.2 Understand the major aetiological factors for substance use disorders, including genetic factors, neurobiological factors, mood and expectancy effects and sociocultural factors.**

A number of aetiological factors have been proposed regarding substance use disorders and some have more support than others. Genetic factors play a role in both alcohol and nicotine dependence. The ability to tolerate alcohol and metabolise nicotine may be what is heritable. Genes crucial to the operation of the dopamine system may be an important factor in explaining how genes influence substance dependence. Several studies show how genes interact with the environment for smoking and alcohol problems. The most-studied neurobiological factors are brain systems associated with dopamine pathways — the major reward pathways in the brain. The incentive-sensitisation theory describes brain pathways involved in liking and wanting (i.e., craving) drugs. People with substance use problems also value immediate rewards more than delayed rewards.

Psychological factors have also been evaluated and there is support for the idea that tension reduction plays a role, but only under certain circumstances, such as when distractions are present. Expectancies about the effects of drugs, such as reducing tension and increasing social skills, have been shown to predict drug and alcohol use. These expectancies are also powerful: the greater the perceived risk of a drug, the less likely it will be used. Studies of personality factors also help us understand why some people may be more prone to abuse drugs and alcohol.

Sociocultural factors play a role, including culture, availability of a substance, family factors, social settings and networks, and advertising. Support exists for both a social influence model and a social selection model.

### **7.3 Describe the approaches to treating substance use disorders, including psychological treatments, medications and drug substitution treatments.**

For heavy alcohol users, withdrawal management is often necessary and may include supportive care and/or medications.

There is some evidence that behavioural couples therapy and motivational interviewing are effective treatments. Controlled drinking refers to a pattern of alcohol consumption that is moderate, avoiding the extremes of total abstinence and inebriation. The guided self-change treatment approach emphasises control over moderate drinking, the costs of drinking to excess and the benefits of abstaining. Self-help programs may also be effective in the treatment of alcohol use disorders. Medications for alcohol use disorder treatment include naltrexone, acamprosate and disulfiram.

Psychological treatments have not been particularly effective for smoking cessation. Scheduled smoking, which involves reducing nicotine intake gradually over a period of a few weeks, has shown some promise. Nicotine gum appears to be somewhat effective, though users can stop chewing the gum. Nicotine patches are more effective than placebo patches, but nine months after the treatment, abstinence differences between those receiving the drug and those receiving a placebo disappear. Adding bupropion or therapy along with nicotine patches may be effective but not for adolescents. Early evidence about e-cigarettes as an effective treatment for stopping smoking is not promising but more work needs to be done.

There is some evidence that CBT is an effective treatment for cocaine dependence. Motivational interviewing has shown promise for the treatment of alcohol and other drug use disorders. Residential treatment homes have not been adequately evaluated for their efficacy, though they are a common form of treatment.

The use of heroin substitutes, such as methadone or naltrexone, is an effective treatment for heroin use disorder. Methadone can only be administered in a special clinic and there is stigma associated with this type of treatment. A prescription drug called buprenorphine can be taken at home. Treating methamphetamine dependence remains a challenge.

### **7.4 Delineate the major approaches to preventing substance use disorders.**

There is evidence to suggest that school-based prevention approaches using social competence and social influence models may be effective in reducing substance use. Psychoeducation alone does not appear effective. Preventions involving parents and families have also been shown to be effective. There is little evidence to suggest that mass media campaigns reduce alcohol and illicit drug use, with some evidence to suggest that they increase illicit drug use among young people. Mass media campaigns have been shown to be effective for smoking.

Alcohol taxation, outlet density and trading hours are all associated with alcohol use. Smoke-free laws, tobacco price increases and televised mass media campaigns have the strongest evidence base in terms of reducing smoking.

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## **KEY TERMS**

**addiction** use of a drug that is accompanied by a physiological dependence on it, made evident by tolerance and withdrawal symptoms

**amphetamines** a group of stimulating drugs that produce heightened levels of energy and, in large doses, nervousness, sleeplessness and paranoid delusions

**caffeine** perhaps the world's most popular drug; a generalised stimulant of body systems, including the sympathetic nervous system. Though seldom viewed as a drug, caffeine is addictive, produces tolerance and subjects habitual users to withdrawal

**cannabis** a drug derived from the dried and ground leaves and stems of the female hemp plant  
*Cannabis sativa*

**cocaine** a pain-reducing, stimulating and addictive alkaloid obtained from coca leaves that increases mental powers, produces euphoria, heightens sexual desire and in large doses causes paranoia and hallucinations

**controlled drinking** a pattern of alcohol consumption that is moderate, avoiding the extremes of total abstinence and of inebriation

**cross-dependent** acting on the same neurotransmitter receptors as methadone does with heroin

**ecstasy** a relatively new hallucinogen, chemically similar to mescaline and the amphetamines

**flashback** an unpredictable recurrence of experiences from an earlier drug high

**foetal alcohol syndrome (FAS)** retarded growth of the developing foetus and infant involving cranial, facial and limb anomalies as well as intellectual disabilities; caused by heavy consumption of alcohol by the mother during pregnancy

**hallucinogen** a drug or chemical, such as LSD, psilocybin or mescaline, whose effects include hallucinations; often called a psychedelic

**hashish** the dried resin of the cannabis plant, stronger in its effects than the dried leaves and stems that constitute cannabis

**heroin** an extremely addictive narcotic drug derived from morphine

**LSD** lysergic acid diethylamide, a drug synthesised in 1938 and discovered by accident to be a hallucinogen in 1943

**MDMA** methylenedioxymethamphetamine, a chemical component of ecstasy; initially used as an appetite suppressant for World War I soldiers and derived from precursors found in nutmeg, dill, saffron and sassafras

**methadone** a synthetic addictive heroin substitute for treating those addicted to heroin that eliminates its effects and the cravings

**methamphetamine** an amphetamine derivative whose abuse skyrocketed in the 1990s

**nicotine** the principal alkaloid of tobacco (an addicting agent)

**nitrous oxide** a gas that, when inhaled, produces euphoria and sometimes giddiness

**opiates** a group of addictive sedatives (including codeine, morphine and oxycodone) that, in moderate doses, relieve pain and induce sleep

**oxycodone** an opiate combined with other drugs to produce prescription pain medications, including the commonly abused drug OxyContin

**secondhand (or passive) smoke** the smoke from the burning end of a cigarette; contains higher concentrations of ammonia, carbon monoxide, nicotine and tar than the smoke inhaled by the smoker

**stimulant** a drug, such as cocaine, that increases alertness and motor activity and at the same time reduces fatigue, allowing an individual to remain awake for an extended period of time

**substance use disorders** disorders in which drugs such as alcohol and cocaine are abused to such an extent that behaviour becomes maladaptive, social and occupational functioning are impaired and control or abstinence becomes impossible. Dependence on the drug may be physiological and produce tolerance and withdrawal

**tolerance** a physiological process in which greater and greater amounts of an addictive drug are required to produce the same effect

**withdrawal** negative physiological and psychological reactions evidenced when a person suddenly stops taking an addictive drug; reactions include cramps, restlessness and even death

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## WEBSITES

1. NHMRC Centre of Research Excellence in Mental Health and Substance Use is an Australian translational research centre that provides evidence-based resources, training and research. ([www.comorbidity.edu.au](http://www.comorbidity.edu.au))
2. National Drug and Alcohol Research Centre is an Australian-based website that contains the latest trends in drug use. ([www.ndarc.med.unsw.edu.au](http://www.ndarc.med.unsw.edu.au))

3. Positive Choices is a portal for teachers, students and parents on drug and alcohol information. ([www.positivechoices.org.au](http://www.positivechoices.org.au))
4. National Drugs Campaign is an Australian government website that provides information about illicit drugs and campaign resources. ([www.australia.gov.au/drugs](http://www.australia.gov.au/drugs))
5. Australian Drug Information Network provides a directory of places in all states and territories in Australia where it is possible to seek information and services to assist with alcohol and other drug related issues. ([www.adin.com.au](http://www.adin.com.au))
6. Drug Info provides information about alcohol and other drugs, as well as an SMS service allowing website users to text the name of a drug to a given number to receive a listing of a number of the drug's effects and links to find further information. ([www.druginfo.adf.org.au](http://www.druginfo.adf.org.au))

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## REFERENCES

- Audrain-McGovern, J., Rodriguez, D., Epstein, L. H., Cuevas, J., Rodgers, K., & Wileyto, E. P. (2009). Does delay discounting play an etiological role in smoking or is it a consequence of smoking? *Drug and Alcohol Dependence*, 103(3), 99–106.
- Australian Institute of Health and Welfare (AIHW). (2014). *National drug household report detailed findings 2013*. Canberra: AIHW.
- Australian Institute of Health and Welfare (AIHW). (2016a). *Alcohol and other drug treatment services in Australia 2014–15*. Canberra: AIHW.
- Australian Institute of Health and Welfare (AIHW). (2016b). *Australian Burden of Disease Study: Impact and causes of illness and deaths in Australia 2011*. Canberra: AIHW.
- Back, S. E., Foa, E., Killeen, T., Mills, K., Teesson, M., Carroll, K. M., & Brady, K. (2014a). *Concurrent treatment for PTSD and substance use using prolonged exposure (COPE): Patient workbook*. New York: Oxford University Press.
- Back, S. E., Foa, E., Killeen, T., Mills, K., Teesson, M., Carroll, K. M., & Brady, K. (2014b). *Concurrent treatment for PTSD and substance use using prolonged exposure (COPE): Therapist manual*. New York: Oxford University Press.
- Ball, J. C., & Ross, A. (1991). *The effectiveness of methadone maintenance treatment*. New York: Springer-Verlag.
- Bechara, A. (2005). Decision making, impulse control and loss of willpower to resist drugs: A neurocognitive perspective. *Nature Neuroscience*, 8(11), 1458–1463.
- Bickel, W. K., Koffarnus, M. N., Moody, L., & Wilson, A. G. (2014). The behavioral- and neuro-economic process of temporal discounting: A candidate behavioral marker of addiction. *Neuropharmacology*, 76, Part B(0), 518–527.
- Bickel, W. K., Miller, M. L., Yi, R., Kowal, B. P., Lindquist, D. M., & Pitcock, J. A. (2007). Behavioral and neuroeconomics of drug addiction: Competing neural systems and temporal discounting processes. *Drug and Alcohol Dependence*, 90 (Suppl. 1), S85–S91.
- Blanch, B., Pearson, S. A., & Haber, P. S. (2014). An overview of the patterns of prescription opioid use, costs and related harms in Australia. *British Journal of Clinical Pharmacology*, 78(5), 1159–1166.
- Boardman, J. D., Saint Onge, J. M., Haberstick, B. C., Timberlake, D. S., & Hewitt, J. K. (2008). Do schools moderate the genetic determinants of smoking? *Behavioral Genetics*, 28, 234–246.
- Bradford, D. E., Shapiro, B. L., & Curtin, J. J. (2013). How bad could it be? Alcohol dampens stress responses to threat of uncertain intensity. *Psychological Science*, 24(12), 2541–2549.
- Brady, K. T., & Sonne, S. C. (1999). The role of stress in alcohol use, alcoholism treatment, and relapse. *Alcohol Research & Health*, 23(4), 263–271.
- Brandon, T. H., Vidrine, J. I., & Litvin, E. B. (2007). Relapse and relapse prevention. *Annual Review of Clinical Psychology*, 3, 257–284.
- Bryden, A., Roberts, B., McKee, M., & Petticrew, M. (2012). A systematic review of the influence on alcohol use of community level availability and marketing of alcohol. *Health and Place*, 18(2), 349–357.
- Budney, A. J., Moore, B. A., Rocha, H. L., & Higgins, S. T. (2006). Clinical trial of abstinence-based vouchers and cognitive behavior therapy for cannabis dependence. *Journal of Consulting and Clinical Psychology*, 74, 307–316.
- Bullen, C., Howe, C., Laugesen, M., McRobbie, H., Parag, V., Williman, J., & Walker, N. (2013). Electronic cigarettes for smoking cessation: a randomised controlled trial. *The Lancet*, 382(9905), 1629–1637.
- Bullers, S., Cooper, M. L., & Russell, M. (2001). Social network drinking and adult alcohol involvement: A longitudinal exploration of the direction of influence. *Addictive Behaviors*, 26, 181–199.
- Burke, B. L., Arkowitz, H., & Menchola, M. (2003). The efficacy of motivational interviewing: A meta-analysis of controlled clinical trials. *Journal of Consulting and Clinical Psychology*, 71, 843–861.
- Burns, L., Breen, C., Bower, C., O'Leary, C., & Elliott, E. J. (2013). Counting fetal alcohol spectrum disorder in Australia: The evidence and the challenges. *Drug and Alcohol Review*, 32(5), 461–467.
- Bushman, B. J., & Cooper, H. M. (1990). Effects of alcohol on human aggression: An integrative research review. *Psychological Bulletin*, 107, 341–354.

- Cahill, K., Stead, L., & Lancaster, T. (2007). Nicotine receptor partial agonists for smoking cessation. *Cochrane Database of Systematic Reviews*, CD006103.
- Camí, J., & Farré, M. (2003). Drug addiction. *New England Journal of Medicine*, 349, 975–986.
- Carroll, K. M., Ball, S. A., Nich, C., et al. (2001). Targeting behavioral therapies to enhance naltrexone treatment of opioid dependence: Efficacy of contingency management and significant other involvement. *Archives of General Psychiatry*, 58, 755–761.
- Carroll, K. M., Easton, C. J., Nich, C., Hunkele, K. A., Neavins, T. M. et al. (2006). The use of contingency management and motivational/skills-building therapy to treat young adults with marijuana dependence. *Journal of Consulting and Clinical Psychology*, 74, 955–966.
- Carroll, K. M., Kiluk, B. D., Nich, C., Gordon, M. A., Portnoy, G. A., Marino, D. R., & Ball, S. A. (2014). Computer-assisted delivery of cognitive-behavioral therapy: Efficacy and durability of CBT4CBT among cocaine-dependent individuals maintained on methadone. *American Journal of Psychiatry*, 171(4), 436–444.
- Carroll, K. M., Rounsaville, B. J., Gordon, L. T., Nich, C., Jatlow, P., Bisighini, R. M., & Gawin, F. H. (1994). Psychotherapy and pharmacotherapy for ambulatory cocaine abusers. *Archives of General Psychiatry*, 51, 177–187.
- Carroll, K. M., Rounsaville, B. J., Nich, C., Gordon, L. T., & Gawin, F. (1995). Integrating psychotherapy and pharmacotherapy for cocaine dependence: Results from a randomized clinical trial. In L. S. Onken, J. D. Blaine & J. J. Boren (Eds.), *Integrating behavioral therapies with medications in the treatment of drug dependence* (pp. 19–36). Rockville, MD: National Institute on Drug Abuse.
- Castells, X., Casas, M., Vidal, X., Bosch, R., Roncero, C., Ramos-Quiroga, J. A., et al. (2007). Efficacy of CNS stimulant treatment for cocaine dependence. A systematic review and meta-analysis of randomized controlled clinical trials. *Addiction*, 102, 1871–1887.
- Caulkins, J. P., Pacula, R. L., Paddock, S., & Chiesa, J. (2004). What we can — and cannot — expect from school-based drug prevention. *Drug and Alcohol Review*, 23(1), 79–87.
- Champion, K. E., Newton, N. C., & Teesson, M. (2016). Prevention of alcohol and other drug use and related harm in the digital age: What does the evidence tell us? *Current Opinion in Psychiatry*, 29(4), 242–249.
- Champion, K. E., Newton, N. C., Barrett, E. L., & Teesson, M. (2013). A systematic review of school-based alcohol and other drug prevention programs facilitated by computers or the Internet. *Drug and Alcohol Review*, 32(2), 115–123.
- Champion, K. E., Newton, N. C., Stapinski, L. A., & Teesson, M. (2016). Effectiveness of a universal internet-based prevention program for ecstasy and new psychoactive substances: a cluster randomized controlled trial. *Addiction*, 111(8), 1396–405. doi: 10.1111/add.13345
- Champion, K. E., Teesson, M., & Newton, N. C. (2015). Patterns and correlates of new psychoactive substance use in a sample of Australian high school students. *Drug and Alcohol Review*. doi: 10.1111/dar.12312
- Chapman, C., Slade, T., Hunt, C., & Teesson, M. (2015). Delay to first treatment contact for alcohol use disorder. *Drug and alcohol dependence*, 147, 116–121.
- Chapman, C., Slade, T., Swift, W., Keyes, K., Tonks, Z., & Teesson, M. (Accepted). Evidence for sex convergence in prevalence of cannabis use: an international systematic review and meta-regression. *Journal of Studies on Alcohol and Drugs*.
- Chassin, L., Curran, P. J., Hussong, A. M., & Colder, C. R. (1996). The relation of parent alcoholism to adolescent substance abuse: A longitudinal follow-up. *Journal of Abnormal Psychology*, 105, 70–80.
- Chassin, L., Pitts, S. C., DeLucia, C., & Todd, M. (1999). A longitudinal study of children of alcoholics: Predicting young adult substance use disorders, anxiety, and depression. *Journal of Abnormal Psychology*, 108, 106–119.
- Chikritzhs, T., Catalano, P., Pascal, R., Henrickson, N. (2007). Predicting alcohol-related harms from licensed outlet density: a feasibility study. *Monograph series* no. 28. Tasmania: NDLERF. Retrieved from [www.ndlerf.gov.au/pub/Monograph\\_28.pdf](http://www.ndlerf.gov.au/pub/Monograph_28.pdf)
- Christakis, N., & Fowler, J. (2008). The collective dynamics of smoking in a large social network. *New England Journal of Medicine*, 358, 2249–2258.
- Cinciripini, P. M., Lapitsky, L. G., Wallfisch, A., Mace, R., Nezami, E., & Van Vunakis, H. (1994). An evaluation of a multicomponent treatment program involving scheduled smoking and relapse prevention procedures: Initial findings. *Addictive Behaviors*, 19, 13–22.
- Cobiac, L., Vos, T., Doran, C., & Wallace, A. (2009). Cost-effectiveness of interventions to prevent alcohol-related disease and injury in Australia. *Addiction*, 104(10), 1646–1655.
- Comer, S. D., Hart, C. L., Ward, A. S., Haney, M., Foltin, R. W., & Fischman, M. W. (2001). Effects of repeated oral methamphetamine administration in humans. *Psychopharmacology*, 155, 397–404.
- Compas, B. E., Haaga, D. A. F., Keefe, F. J., Leitenberg, H., & Williams, D. A. (1998). Sampling of empirically supported psychological treatments from health psychology: Smoking, chronic pain, cancer, and bulimia nervosa. *Journal of Consulting and Clinical Psychology*, 66, 89–112.
- Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotion: A motivational model of alcoholism. *Journal of Personality and Social Psychology*, 69, 961–974.
- Curry, S. J., Mermelstein, R. J., & Sporer, A. K. (2009). Therapy for specific problems: Youth tobacco cessation. *Annual Review of Psychology*, 60, 229–255.
- Curtin, J. J., Lang, A. R., Patrick, C. J., & Strizke, W. G. K. (1998). Alcohol and fear-potentiated startle: The role of competing cognitive demands in the stress-reducing effects of intoxication. *Journal of Abnormal Psychology*, 107, 547–557.
- Dallery, J., Silverman, K., Chutuape, M. A., Bigelow, G. E., & Stitzer, M. (2001). Voucher-based reinforcement of opiate plus cocaine abstinence in treatment-resistant methadone patients: Effects of reinforcer magnitude. *Experimental and Clinical Psychopharmacology*, 9, 317–325.

- Dalton, M. A., Sargent, J. D., Beach, M. L., Titus-Ernstoff, L., Gibson, J. J., Ahrens, M. B., ... Heatherton, T. F. (2003). Effect of viewing smoking in movies on adolescent smoking initiation: A cohort study. *The Lancet*, 362(9380), 281–285.
- Darke, S., Marel, C., Mills, K. L., Ross, J., Slade, T., Burns, L., & Teesson, M. (2014). Patterns and correlates of non-fatal heroin overdose at 11-year follow-up: Findings from the Australian Treatment Outcome Study. *Drug and Alcohol Dependence*, 144, 148–152.
- Daube, M., Eastwood, P., Mishima, M., & Peters, M. (2015). Tobacco plain packaging: The Australian experience. *Respirology*, 20(7), 1001–1003.
- de Wit, H., & Zacny, J. (2000). Abuse potential of nicotine replacement therapies. In K. J. Palmer (Ed.), *Smoking Cessation* (pp. 79–92). Kwai Chung, Hong Kong: Adis International Publications.
- Deady, M., Mills, K. L., Teesson, M., & Kay-Lambkin, F. (2016). An online intervention for co-occurring depression and problematic alcohol use in young people: Primary outcomes from a randomized controlled trial. *Journal of Medical Internet Research*, 18(3).
- Degenhardt, L., Bruno, R., Ali, R., Lintzeris, N., Farrell, M., & Larance, B. (2015). The introduction of a potentially abuse deterrent oxycodone formulation: Early findings from the Australian National Opioid Medications Abuse Deterrence (NOMAD) study. *Drug and alcohol dependence*, 151, 56–67.
- Degenhardt, L., Coffey, C., Carlin, J. B., Swift, W., Moore, E., & Patton, G. C. (2010). Outcomes of occasional cannabis use in adolescence: 10-year follow-up study in Victoria, Australia. *The British Journal of Psychiatry*, 196(4), 290–295.
- Degenhardt, L., Larney, S., Chan, G., Dobbins, T., Weier, M., Roxburgh, A., ... McKetin, R. (2016). Estimating the number of regular and dependent methamphetamine users in Australia, 2002–2014. *Medical Journal of Australia*, 204(4), 153.
- Dick, D. M., Pagan, J. L., Viken, R., Purcell, S., Kaprio, J., Pulkkinen, L., et al. (2007). Changing environmental influences on substance use across development. *Twin Research and Human Genetics*, 10, 315–326.
- Elder, R. W., Lawrence, B., Ferguson, A., Naimi, T. S., Brewer, R. D., Chattopadhyay, S. K., ... Services, T. F. o. C. P. (2010). The effectiveness of tax policy interventions for reducing excessive alcohol consumption and related harms. *American Journal of Preventive Medicine*, 38(2), 217–229.
- Elliott, E. J., Payne, J., Morris, A., Haan, E., & Bower, C. (2008). Fetal alcohol syndrome: A prospective national surveillance study. *Archives of Disease in Childhood*, 93(9), 732–737.
- Etter, J. F., & Bullen, C. (2014). A longitudinal study of electronic cigarette users. *Addictive Behaviors*, 39(2), 491–494.
- Faggiano, F., Minozzi, S., Versino, E., & Buscemi, D. (2013). Universal school-based prevention for illicit drug use. *The Cochrane Database of Systematic Reviews*, 12, CD003020-CD003020.
- Fals-Stewart, W., O'Farrell, T. J., & Lam, W. K. K. (2009). Behavioral couple therapy for gay and lesbian couples with alcohol use disorders. *Journal of Substance Abuse Treatment*, 37(4), 379–387.
- Ferguson, S. B., Shiffman, S., & Gwaltney, C. J. (2006). Does reducing withdrawal severity mediate nicotine patch efficacy? A randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 74, 1153–1161.
- Ferri, M., Amato, L., & Davoli, M. (2008). Alcoholics anonymous and other 12 step programmes for alcohol dependence (review). *Cochrane Database of Systematic Reviews*, Issue 3. Art. No.: CD005032.
- Fiellin, D. A., O'Connor, P. G., Chawarski, M., Pakes, J. P., Pantalon, M. V., & Schottenfeld, R. S. (2001). Methadone maintenance in primary care: A randomized controlled trial. *Journal of the American Medical Association*, 286, 1724–1731.
- Fiorentine, R., & Hillhouse, M. P. (2000). Exploring the additive effects of drug misuse treatment and Twelve Step involvement: Does Twelve-Step ideology matter? *Substance Use and Misuse*, 35, 367–397.
- Foxcroft, D. R., & Tsertsvadze, A. (2011). Universal family-based prevention programs for alcohol misuse in young people. *Cochrane Database of Systematic Reviews*, 9, CD009308.
- Foxcroft, D. R., Coombes, L., Wood, S., Allen, D., & Almeida Santimano, N. M. (2014). Motivational interviewing for alcohol misuse in young adults. *The Cochrane Library*.
- Frazer, K., Callinan, J. E., McHugh, J., van Baarsel, S., Clarke, A., Doherty, K., & Kelleher, C. (2016). Legislative smoking bans for reducing harms from secondhand smoke exposure, smoking prevalence and tobacco consumption. *Cochrane Database of Systematic Reviews*, 2. doi: 10.1002/14651858.CD005992.pub3
- Frost, D. O., & Cadet, J.-L. (2000). Effects of methamphetamine-induced toxicity on the development of neural circuitry: A hypothesis. *Brain Research Reviews*, 34, 103–118.
- Gibson, D. R. (2001). Effectiveness of syringe exchange programs in reducing HIV risk behavior and seroconversion among injecting drug users. *AIDS*, 15, 1329–1341.
- Grana, R. A., Popova, L., & Ling, P. M. (2014). A longitudinal analysis of electronic cigarette use and smoking cessation. *Journal of the American Medical Association, Internal Medicine*, 174(5), 812–813.
- Grinspoon, L., & Bakalar, J. B. (1995). Marijuana as medicine: A plea for reconsideration. *Journal of the American Medical Association*, 273, 1875–1876.
- Haber, P., Lintzeris, N., Proude, E., & Lopatko, O. (2009). *Guidelines for the treatment of alcohol problems*. Canberra: Department of Health and Ageing.
- Hahn, R. A., Kuzara, J. L., Elder, R., Brewer, R., Chattopadhyay, S., Fielding, J., ... Lawrence, B. (2010). Effectiveness of policies restricting hours of alcohol sales in preventing excessive alcohol consumption and related harms. *American Journal of Preventive Medicine*, 39(6), 590–604.
- Hall, W. D., Ross, J. E., Lynskey, M. T., Law, M. G., & Degenhardt, L. J. (2000). How many dependent heroin users are there in Australia? *The Medical Journal of Australia*, 173(10), 528–531.
- Hammond, D. (2011). Health warning messages on tobacco products: Review. *Tobacco Control*, 20(5), 327–337.

- Harden, K. P., Hill, J. E., Turkheimer, E., & Emery, R. E. (2008). Gene–environment correlation and interaction in peer effects on adolescent alcohol and tobacco use. *Behavior Genetics*, 38, 339–347.
- Hartz, D. T., Fredrick-Osborne, S. L., & Galloway, G. P. (2001). Craving predicts use during treatment for methamphetamine dependence: A prospective repeated measures, within-subjects analysis. *Drug and Alcohol Dependence*, 63, 269–276.
- Hasin, D. S., O'Brien, C. P., Auriacombe, M., Borges, G., Bucholz, K., Budney, A., et al. (2013). DSM-5 criteria for substance use disorders: Recommendations and rationale. *American Journal of Psychiatry*, 170(8), 834–851.
- Hettema, J. E., & Hendricks, P. S. (2010). Motivational interviewing for smoking cessation: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, 78(6), 868–884.
- Huang, J., Chaloupka, F. J., & Fong, G. T. (2014). Cigarette graphic warning labels and smoking prevalence in Canada: A critical examination and reformulation of the FDA regulatory impact analysis. *Tobacco Control*, 23 Suppl 1, i7–12.
- Huether, G., Zhou, D., & Ruther, E. (1997). Causes and consequences of the loss of serotonergic presynapses elicited by the consumption of 3,4-ethylenedioxymethamphetamine (MDMA, “ecstasy”) and its congeners. *Journal of Neural Transmission*, 104, 771–794.
- Hughes, J. R. (2009). How confident should we be that smoking cessation treatments work? *Addiction*, 104(10), 1637–1640.
- Hughes, J. R., Higgins, S. T., & Hatsukami, D. K. (1990). Effects of abstinence from tobacco: A critical review. In L. T. Kozlowski, H. Annis, H. D. Cappell, F. Glaser, M. Goodstadt, Y. Israel, H. Kalant, E. M. Sellers & J. Vingilis (Eds.), *Research advances in alcohol and drug problems*. New York: Plenum.
- Hughes, J. R., Higgins, S. T., Bickel, W. K., Hunt, W. K., & Fenwick, J. W. (1991). Caffeine self-administration, withdrawal, and adverse effects among coffee drinkers. *Archives of General Psychiatry*, 48, 611–617.
- Hughes, J., Stead, L., & Lancaster, T. (2004). Antidepressants for smoking cessation. *Cochrane Database of Systematic Reviews*, CD000031.
- Hussong, A. M., Hicks, R. E., Levy, S. A., & Curran, P. J. (2001). Specifying the relations between affect and heavy alcohol use among young adults. *Journal of Abnormal Psychology*, 110, 449–461.
- Institute of Medicine. (1990). *Treating drug problems*. Washington, DC: National Academy Press.
- Intergovernmental Committee on Drugs. (2012). *National tobacco strategy 2012–2018*. Canberra: Commonwealth of Australia.
- Ito, T., Miller, N., & Pollock, V. (1996). Alcohol and aggression: A meta-analysis on the moderating effects of inhibitory cues, triggering events, and self-focused attention. *Psychological Bulletin*, 120, 60–82.
- Jaffe, J. H. (1985). Drug addiction and drug abuse. In Goodman, A. G. & Gilman, A. (Eds.), *Goodman and Gilman's The pharmacological basis of therapeutic behavior*. New York: Macmillan.
- Johnson, B. A., Rosenthal, N., Capece, J. A., Wiegand, F., Mao, L., Beyers, K., ... Anton, R. F. (2008). Improvement of physical health and quality of life of alcohol-dependent individuals with topiramate treatment: US multisite randomized controlled trial. *Archives of Internal Medicine*, 168(11), 1188–1199.
- Johnson, B. A., Rosenthal, N., Capece, J. A., Wiegand, F., Mao, L., Beyers, K., ... Ciraulo, D. A. (2007). Topiramate for treating alcohol dependence: A randomized controlled trial. *JAMA*, 298(14), 1641–1651.
- Johnston, L. D., O'Malley, P. M., Bachman, J. G., Schulenberg, J. E., & Miech, R. A. (2014). Monitoring the future national survey results on drug use, 1975–2013: Volume I, Secondary school. Ann Arbor: Institute for Social Research, The University of Michigan.
- Jorenby, D. E., Leischow, S. J., Nides, M. A., Rennard, S. I., Johnston, J. A., et al. (1999). A controlled trial of sustained-release bupropion, a nicotine patch, or both for smoking cessation. *New England Journal of Medicine*, 340, 685–691.
- Josephs, R. A., & Steele, C. M. (1990). The two faces of alcohol myopia: Attentional mediation of psychological stress. *Journal of Abnormal Psychology*, 99, 115–126.
- Kassel, J. D., & Shiffman, S. (1997). Attentional mediation of cigarette smoking's effect on anxiety. *Health Psychology*, 16, 359–368.
- Kassel, J. D., & Unrod, M. (2000). Smoking, anxiety, and attention: Support for the role of nicotine in attentionally mediated anxiety. *Journal of Abnormal Psychology*, 109, 161–166.
- Kassel, J. D., Stroud, L. R., & Paronis, C. A. (2003). Smoking, stress, and negative affect: Correlation, causation, and context across stages of smoking. *Psychological Bulletin*, 129, 270–304.
- Katz, E. C., Gruber, K., Chutuape, M. A., & Stitzer, M. L. (2001). Reinforcement-based outpatient treatment for opiate and cocaine abusers. *Journal of Substance Abuse Treatment*, 20, 93–98.
- Kendler, K. S., & Prescott, C. A. (1998). Cannabis use, abuse, and dependence in a population-based sample of female twins. *American Journal of Psychiatry*, 155, 1016–1022.
- Kendler, K. S., Chen, X., Dick, D., Maes, H., Gillespie, N., Neale, M. C., & Riley, B. (2012). Recent advances in the genetic epidemiology and molecular genetics of substance use disorders. *Nature Neuroscience*, 15(2), 181–189.
- Kendler, K. S., Jacobson, K. C., Prescott, C. A., & Neale, M. C. (2003). Specificity of genetic and environmental risk factors for use and abuse/dependence of cannabis, cocaine, hallucinogens, sedatives, stimulants, and opiates in male twins. *American Journal of Psychiatry*, 160, 687–695.
- Kendler, K. S., Myers, J., & Prescott, C. A. (2007). Specificity of genetic and environmental risk factors for symptoms of cannabis, cocaine, alcohol, caffeine and nicotine dependence. *Archives of General Psychiatry*, 64(11), 1313–1320.
- Kendler, K. S., Prescott, C. A., Myers, J., & Neale, M. C. (2003). The structure of genetic and environmental risk factors for common psychiatric and substance use disorders in men and women. *Archives of General Psychiatry*, 60, 929–937.
- Khadjesari, Z., Murray, E., Hewitt, C., Hartley, S., & Godfrey, C. (2011). Can stand-alone computer-based interventions reduce alcohol consumption? A systematic review. *Addiction*, 106(2), 267–282.

- Killen, J. D., Fortmann, S. P., Murphy Jr., G. M., Hayward, C., Arredondo, C. et al. (2006). Extended treatment with bupropion SR for cigarette smoking cessation. *Journal of Consulting and Clinical Psychology*, 74, 286–294.
- Kiluk, B. D., Sugarman, D. E., Nich, C., Gibbons, C. J., Martino, S., Rounsaville, B. J., & Carroll, K. M. (2011). A methodological analysis of randomized clinical trials of computer-assisted therapies for psychiatric disorders: Toward improved standards for an emerging field. *American Journal of Psychiatry*, 168(8), 790–9. doi: 10.1176/appi.ajp.2011.10101443
- King, A. C., McNamara, P. J., Hasin, D. S., & Cao, D. (2014). Alcohol challenge responses predict future alcohol use disorder symptoms: A 6-year prospective study. *Biological Psychiatry*, 75(10), 798–806.
- Kloner, R. A., & Rezkalla, S. H. (2007). To drink or not to drink? That is the question. *Circulation*, 116, 1306–1317.
- Knott, C. S., Coombs, N., Stamatakis, E., & Biddulph, J. P. (2015). All cause mortality and the case for age specific alcohol consumption guidelines: Pooled analyses of up to 10 population based cohorts. *BMJ*, 350, h384. doi: <https://doi.org/10.1136/bmj.h384>
- Koob, G. F., Caine, B., Hyttia, P., Markou, A., Parsons, L. H., Roberts, A. J., et al. (1999). Neurobiology of drug addiction. In M. D. Glantz & C. R. Hartel (Eds.), *Drug abuse: Origins and interventions* (pp. 161–190). Washington, DC: American Psychological Association.
- Koob, G. F. (2008). A role for brain systems in addiction. *Neuron*, 59, 11–34.
- Koob, G. F., & Le Moal. (2008). Addiction and the brain antireward system. *Annual Review of Psychology*, 59, 29–53.
- Kranzler, H. R., & van Kirk, J. (2001). Efficacy of naltrexone and acamprosate for alcoholism treatment: A metaanalysis. *Alcoholism: Clinical and Experimental Research*, 25, 1335–1341.
- Krystal, J. H., Cramer, J. A., Krol, W. F., et al. (2001). Naltrexone in the treatment of alcohol dependence. *New England Journal of Medicine*, 345, 1734–1739.
- Lai, D. T., Cahill, K., Qin, Y., & Tang, J. L. (2010). Motivational interviewing for smoking cessation. *Cochrane Database of Systematic Reviews*(1), CD006936.
- Lang, A. R., Goeckner, D. J., Adessor, V. J., & Marlatt, G. A. (1975). Effects of alcohol on aggression in male social drinkers. *Journal of Abnormal Psychology*, 84, 508–518.
- Law, M., & Tang, J. L. (1995). An analysis of the effectiveness of interventions intended to help people stop smoking. *Archives of Internal Medicine*, 155, 1933–1941.
- Leeman, R. F., Perez, E., Nogueira, C., & DeMartini, K. S. (2015). Very-brief, web-based interventions for reducing alcohol use and related problems among college students: A review. *Frontiers in Psychiatry*, 6 (SEP) (129).
- Liechti, M. E., Baumann, C., Gamma, A., & Vollenweider, F. X. (2000). Acute psychological effects of 3, 4-methylenedioxymethamphetamine (MDMA, “Ecstasy”) are attenuated by the serotonin uptake inhibitor citalopram. *Neuropsychopharmacology*, 22, 513–521.
- Lilly, R., Quirk, A., Rhodes, T., & Stimson, G. V. (2000). Sociality in methadone treatment: Understanding methadone treatment and service delivery as a social process. *Drugs: Education, Prevention and Policy*, 7, 163–178.
- Lopez, A. D. (2006). *Global burden of disease and risk factors*. Washington: World Bank Publications.
- Marlatt, G. A. (1983). The controlled drinking controversy: A commentary. *American Psychologist*, 38, 1097–1110.
- Marlatt, G. A., & Gordon, J. R. (Eds.). (1985). *Relapse prevention: Maintenance strategies in the treatment of addictive behaviors*. New York: Guilford Press.
- Martell, B. A., Orson, F. M., Poling, J., Mitchell, E., Rossen, R. D., Gardner, T., & Kosten, T. R. (2009). Cocaine vaccine for the treatment of cocaine dependence in methadone-maintained patients: A randomized, double-blind, placebo-controlled efficacy trial. *Archives of General Psychiatry*, 66, 1116–1123.
- Mason, B. J. (2001). Treatment of alcohol-dependent outpatients with acamprosate: A clinical review. *Journal of Clinical Psychiatry*, 62, 42–48.
- Matsuda, L. A., Lolait, S. J., Brownstein, M. J., Young, A. C., & Bonner, T. I. (1990). Structure of a cannabinoid receptor and functional expression of the cloned cDNA. *Nature*, 346, 561–564.
- McCready, B. S., & Epstein, E. E. (1995). Directions for research on alcoholic relationships: Marital and individual-based models of heterogeneity. *Psychology of Addictive Behaviors*, 9, 157–166.
- McGue, M., Pickens, R. W., & Sviki, D. S. (1992). Sex and age effects on the inheritance of alcohol problems: A twin study. *Journal of Abnormal Psychology*, 101, 3–17.
- McKetin, R., Najman, J. M., Baker, A. L., Lubman, D. I., Dawe, S., Ali, R., . . . Mamun, A. (2012). Evaluating the impact of community-based treatment options on methamphetamine use: Findings from the Methamphetamine Treatment Evaluation Study (MATES). *Addiction*, 107(11), 1998–2008.
- McLaren, J., Swift, W., Dillon, P., & Allsop, S. (2008). Cannabis potency and contamination: A review of the literature. *Addiction*, 103(7), 1100–1109.
- Meier, M. H., Caspi, A., Ambler, A., Harrington, H., Houts, R., Keefe, R. S. E., et al. (2012). Persistent cannabis users show neuropsychological decline from childhood to midlife. *Proceedings of the National Academy of Sciences*, 109(40), E2657–E2664.
- Mewton, L., Teesson, M., Slade, T., & Grove, R. (2011). The epidemiology of DSM-IV alcohol use disorders amongst young adults in the Australian population. *Alcohol and Alcoholism*, 46(2), 185–191.
- Mills, K. L., Teesson, M., Back, S. E., Brady, K. T., Baker, A. L., Hopwood, S., . . . Rosenfeld, J. (2012). Integrated exposure-based therapy for co-occurring posttraumatic stress disorder and substance dependence: a randomized controlled trial. *JAMA*, 308(7), 690–699.
- Morgan, M. J. (2000). Ecstasy (MDMA): A review of its possible persistent psychological effects. *Psychopharmacology*, 152, 230–248.

- Munro, S., Thomas, K. L., & Abu-Shaar, M. (1993). Molecular characterization of a peripheral receptor for cannabinoids. *Nature*, 365, 61–65.
- National Centre in HIV Epidemiology and Clinical Research. (2009). *Return on investment 2: Evaluating the cost-effectiveness of needle and syringe programs in Australia*. Canberra: Author.
- Newton, N. C., Barrett, E. L., Castellanos-Ryan, N., Kelly, E., Champion, K. E., Stapinski, L., ... Teesson, M. (2016). The validity of the Substance Use Risk Profile Scale (SURPS) among Australian adolescents. *Addictive Behaviours*, 53, 23–30.
- Newton, N. C., Conrod, P. J., Slade, T., Carragher, N., Champion, K. E., Barrett, E. L., ... Teesson, M. (2016). The long-term effectiveness of a selective, personality-targeted prevention program in reducing alcohol use and related harms: a cluster randomized controlled trial. *Journal of Child Psychology and Psychiatry*, 57(9), 1056–65. doi: 10.1111/jcpp.12558.
- Nielsen, S., Roxburgh, A., Bruno, R., Lintzeris, N., Jefferson, A., & Degenhardt, L. (2015). Changes in non-opioid substitution treatment episodes for pharmaceutical opioids and heroin from 2002 to 2011. *Drug and Alcohol Dependence*, 149, 212–219.
- O'Farrell, T. J., & Fals-Stewart, W. (2000). Behavioral couples therapy for alcoholism and drug abuse. *Journal of Substance Abuse Treatment*, 18, 51–54.
- Offord, D. R., & Kraemer, H. C. (2000). Risk factors and prevention. *Evidence Based Mental Health*, 3(3), 70–71.
- Oscar-Berman, M., & Marinković, K. (2007). Alcohol: Effects on neurobehavioural functions and the brain. *Neuropsychology review*, 17(3), 239–257.
- Paulus, M. P., Tapert, S. F. & Schuckit, M. A. (2005). Neural activation patterns of methamphetamine-dependent subjects during decision making predict relapse. *Archives of General Psychiatry*, 62, 761–768.
- Perkins, K. A., Ciccocioppo, M., Conklin, C. A., Milanek, M. E., Grotenthaler, A., & Sayette, M. A. (2008). Mood influences on acute smoking responses are independent of nicotine intake and dose expectancy. *Journal of Abnormal Psychology*, 117, 79–93.
- Perkins, K. A., Karelitz, J. L., Conklin, C. A., Sayette, M. A., & Giedgowd, G. E. (2010). Acute negative affect relief from smoking depends on the affect situation and measure but not on nicotine. *Biological Psychiatry*, 67, 707–714.
- Petrie, J., Bunn, F., & Byrne, G. (2007). Parenting programmes for preventing tobacco, alcohol or drugs misuse in children <18: A systematic review. *Health Education Research*, 22(2), 177–191.
- Petry, N. M., Alessi, S. M., & Hanson. (2007). Contingency management improves abstinence and quality of life in cocaine abusers. *Journal of Consulting and Clinical Psychology*, 75, 307–315.
- Petry, N. M., Alessi, S. M., Marx, J., Austin, M., & Tardif, M. (2005). Vouchers versus prizes: Contingency management treatment of substance abusers in community settings. *Journal of Consulting and Clinical Psychology*, 73, 1005–1014.
- Pettinati, H. M., Oslin, D. W., Kampman, K. M., Dundon, W. D., Xie, H., Gallis, T. L., et al. (2010). A double-blind, placebo-controlled trial combining sertraline and naltrexone for treating co-occurring depression and alcohol dependence. *American Journal of Psychiatry*, 167, 668–675.
- Pierce, J. M., Petry, N. M., Stitzer, M. L., Blaine, J., Kellog, S., et al. (2006). Effects of lower-cost incentives on stimulant abstinence in methadone maintenance treatment. *Archives of General Psychiatry*, 63, 201–208.
- Polich, J. M., Armor, D. J., & Braiker, H. B. (1980). Patterns of alcoholism over four years. *Journal of Studies on Alcohol*, 41, 397–415.
- Popova, S., Giesbrecht, N., Bekmuradov, D., & Patra, J. (2009). Hours and days of sale and density of alcohol outlets: impacts on alcohol consumption and damage: a systematic review. *Alcohol and Alcoholism*, 44(5), 500–516.
- Powers, M. B., Vedel, E., & Emmelkamp, P. M. G. (2008). Behavioral couples therapy (BCT) for alcohol and drug use disorders: A meta-analysis. *Clinical Psychology Review*, 28(6), 952–962.
- Rando, K., Chaplin, T. M., Potenza, M. N., Mayes, L., & Sinha, R. (2013). Prenatal cocaine exposure and gray matter volume in adolescent boys and girls: Relationship to substance use initiation. *Biological Psychiatry*, 74(7), 482–489.
- Rather, B. C., Goldman, M. S., Roehrich, L., & Brannick, M. (1992). Empirical modeling of an alcohol expectancy memory network using multidimensional scaling. *Journal of Abnormal Psychology*, 101, 174–183.
- Rawson, R. A., Martinelli-Casey, P., Anglin, M. D., et al. (2004). A multi-site comparison of psychosocial approaches for the treatment of methamphetamine dependence. *Addiction*, 99, 708–717.
- Richardson, K., Baillie, A., Reid, S., Morley, K., Teesson, M., Sannibale, C., ... Haber, P. (2008). Do acamprosate or naltrexone have an effect on daily drinking by reducing craving for alcohol? *Addiction*, 103(6), 953–959.
- Robbins, S. J., Ehrman, R. N., Childress, A. R., Cornish, J. W., & O'Brien, C. P. (2000). Mood state and recent cocaine use are not associated with levels of cocaine cue reactivity. *Drug and Alcohol Dependence*, 59, 33–42.
- Roberts, C. A., Jones, A., & Montgomery, C. (2016). Meta-analysis of molecular imaging of serotonin transporters in ecstasy/polydrug users. *Neuroscience & Biobehavioural Reviews*, 63, 158–167.
- Robinson, T. E., & Berridge, K. C. (1993). The neural basis of drug craving: An incentive sensitization theory of addiction. *Brain Research Reviews*, 18, 247–191.
- Robinson, T. E., & Berridge, K. C. (2003). Addiction. *Annual Review of Psychology*, 54, 25–53.
- Rogers, P. J., Heatherley, S. V., Mullings, E. L., & Smith, J. E. (2013). Faster but not smarter: Effects of caffeine and caffeine withdrawal on alertness and performance. *Psychopharmacology*, 226(2), 229–240.
- Rooke, S., Thorsteinsson, E., Karpin, A., Copeland, J., & Allsop, D. (2010). Computer-delivered interventions for alcohol and tobacco use: a meta-analysis. *Addiction*, 105(8), 1381–1390.
- Rose, J. E., & Behm, F. M. (2014). Combination Treatment With Varenicline and Bupropion in an Adaptive Smoking Cessation Paradigm. *Goldman Journal of Psychiatry*.

- Rose, J. E., Herskovic, J. E., Behm, F. M., & Westman, E. C. (2009). Precessation treatment with nicotine patch significantly increases abstinence rates relative to conventional treatment. *Nicotine and Tobacco Research*, 11(9), 1067–1075.
- Ross, J., Teesson, M., Lejuez, C., Mills, K., Kaye, S., Brady, K., . . . Cassar, J. (2016). The efficacy of behavioural activation treatment for co-occurring depression and substance use disorder (the activate study): A randomized controlled trial. *BMC Psychiatry*, 16(1), 221.
- Ryan, S. M., Jorm, A. F., & Lubman, D. I. (2010). Parenting factors associated with reduced adolescent alcohol use: A systematic review of longitudinal studies. *Australian and New Zealand Journal of Psychiatry*, 44(9), 774–783.
- Sacco, R. L., Elkind, M., Boden-Albala, B., Lin, I., Kargman, D. E., et al. (1999). The protective effect of moderate alcohol consumption on ischemic stroke. *Journal of the American Medical Association*, 281, 53–60.
- Schumacher, J. E., Milby, J. B., Wallace, D., Meehan, D. C., Kertesz, S. et al. (2007). Meta-analysis of day treatment and contingency-management dismantling research: Birmingham homeless cocaine studies (1990–2005). *Journal of Consulting and Clinical Psychology*, 75, 823–828.
- Sher, K. J., Walitzer, K. S., Wood, P. K., & Brent, E. F. (1991). Characteristics of children of alcoholics: Putative risk factors, substance use and abuse, and psychopathology. *Journal of Abnormal Psychology*, 100, 427–448.
- Sher, K. J., Wood, M. D., Wood, P. K., & Raskin, G. (1996). Alcohol outcome expectancies and alcohol use: A latent variable cross-lagged panel study. *Journal of Abnormal Psychology*, 105, 561–574.
- Shiffman, S., & Waters, A. J. (2004). Negative affect and smoking lapses: A prospective analysis. *Journal of Consulting and Clinical Psychology*, 72, 192–201.
- Shiffman, S., Gwaltney, C. J., Balabanis, M. H., Liu, K. S., Paty, J. A., Kassel, J. D., Hickcox, M., & Gnys, M. (2002). Immediate antecedents of cigarette smoking: An analysis from ecological momentary assessment. *Journal of Abnormal Psychology*, 111, 531–545.
- Shiffman, S., Paty, J. A., Gwaltney, C. J., & Dang, Q. (2004). Immediate antecedents of cigarette smoking: An analysis of unrestricted smoking patterns. *Journal of Abnormal Psychology*, 113, 166–171.
- Silverman, K., Higgins, S. T., Brooner, R. K., Montoya, I. D., Cone, E. J., Schuster, C. R., & Preston, K. I. (1996). Sustained cocaine abstinence in methadone maintenance patients through voucher-based reinforcement therapy. *Archives of General Psychiatry*, 53, 409–413.
- Slade, T., Chapman, C., Swift, W., Keyes, K., Tonks, Z., & Teesson, M. (2016). Birth cohort trends in the global epidemiology of alcohol use and alcohol-related harms in men and women: systematic review and metaregression. *BMJ open*, 6(10), e011827.
- Slade, T., Johnston, A., Oakley Browne, M. A., Andrews, G., & Whiteford, H. (2009). 2007 National Survey of Mental Health and Wellbeing: Methods and key findings. *Australian and New Zealand Journal of Psychiatry*, 43(7), 594–605.
- Smart, R. G., & Ogburne, A. C. (2000). Drug use and drinking among students in 36 countries. *Addictive Behaviors*, 25, 455–460.
- Smith, G. T., Goldman, M. S., Greenbaum, P. E., & Christiansen, B. A. (1995). Expectancy for social facilitation from drinking: The divergent paths of high expectancy and low expectancy adolescents. *Journal of Abnormal Psychology*, 104, 32–40.
- Sobell, L. C., Sobell, M. B., & Agrawal, S. (2009). Randomized controlled trial of a cognitive-behavioral motivational intervention in a group versus individual format for substance use disorders. *Psychology of Addictive Behaviors: Journal of the Society of Psychologists in Addictive Behaviors*, 23, 672–683.
- Sobell, L. C., Toneatto, A., & Sobell, M. B. (1990). Behavior therapy. In A. S. Bellack & M. Hersen (Eds.), *Handbook of comparative treatments for adult disorders* (pp. 479–505). New York: John Wiley & Sons.
- Sobell, M. B., & Sobell, L. C. (1993). *Problem drinkers: Guided self-change treatment*. New York: Guilford Press.
- Stacy, A. W., Newcomb, M. D., & Bentler, P. M. (1991). Cognitive motivation and drug use: A 9-year longitudinal study. *Journal of Abnormal Psychology*, 100, 502–515.
- Stafford J., Sutherland, R., Burns, L., & Breen, C (2016). The 2016 EDRS key findings: a survey of people who regularly use psychostimulant drugs. *EDRS Drug Trends Bulletin*, October 2016. Sydney: National Drug and Alcohol Research Centre, University of New South Wales.
- Stead, L. F., Perera, R., Bullen, C., Mant, D., & Lancaster, T. (2008). Nicotine replacement therapy for smoking cessation. *Cochrane Database of Systematic Reviews*, No.: CD000146.
- Steele, C. M., & Josephs, R. A. (1988). Drinking your troubles away: 2. An attention-allocation model of alcohol's effects on psychological stress. *Journal of Abnormal Psychology*, 97, 196–205.
- Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia: Its prized and dangerous effects. *American Psychologist*, 45(8), 921–933.
- Stice, E., Barrera, M., & Chasin, L. (1998). Prospective differential prediction of adolescent alcohol use and problem use: Examining the mechanisms of effect. *Journal of Abnormal Psychology*, 107, 616–628.
- Stockings, E., Hall, W. D., Lynskey, M., Morley, K. I., Reavley, N., Strang, J., . . . Degenhardt, L. (2016). Prevention, early intervention, harm reduction and treatment of substance use in young people. *The Lancet Psychiatry*, 3(3), 280–296.
- Strain, E. C., Bigelow, G. E., Liebson, I. A., & Stitzer, M. L. (1999). Moderate- vs low-dose methadone in the treatment of opioid dependence. *Journal of the American Medical Association*, 281, 1000–1005.
- Streton, C., & Whelan, G. (2001). Naltrexone, a relapse prevention maintenance treatment of alcohol dependence: A meta-analysis of randomized controlled trials. *Alcohol and Alcoholism*, 36, 544–552.
- Stritzke, W. G. K., Patrick, C. J., & Lang, P. J. (1995). Alcohol and emotion: A multidimensional approach incorporating startle probe methodology. *Journal of Abnormal Psychology*, 104, 114–122.
- Substance Abuse and Mental Health Services Administration (SAMHSA). (2013). Drug Abuse Warning Network, 2011: National Estimates of Drug-Related Emergency Department Visits. HHS Publication No. (SMA) 13-4760, DAWN Series D-39. Rockville, MD: Substance Abuse and Mental Health Services Administration.

- Sullivan, J. M. (2000). Cellular and molecular mechanisms underlying learning and memory impairments produced by cannabinoids. *Learning and Memory*, 7, 132–139.
- Sussman, S., Dent, C. W., McAdams, L., Stacy, A. W., Burton, D., & Flay, B. R. (1994). Group self-identification and adolescent cigarette smoking: A 1-year prospective study. *Journal of Abnormal Psychology*, 103, 576–580.
- Sussman, S., Stacy, A. W., Dent, C. W., Simon, T. R., & Johnson, C. A. (1996). Marijuana use: Current issues and new research directions. *Journal of Drug Issues*, 26, 695–733.
- Swisher, J. D., Scherer, J., & Yin, R. K. (2004). Cost-benefit estimates in prevention research. *Journal of Primary Prevention*, 25(2), 137–148.
- Tait, R. J., Spijkerman, R., & Riper, H. (2013). Internet and computer based interventions for cannabis use: A meta-analysis. *Drug and Alcohol Dependence*, 133(2), 295–304.
- Teesson, M., Hall, W., Slade, T., Mills, K., Grove, R., Mewton, L., ... Haber, P. (2010). Prevalence and correlates of DSM-IV alcohol abuse and dependence in Australia: Findings of the 2007 National Survey of Mental Health and Wellbeing. *Addiction*, 105(12), 2085–2094.
- Teesson, M., Marel, C., Darke, S., Ross, J., Slade, T., Burns, L., ... Mills, K. L. (2015). Long-term mortality, remission, criminality and psychiatric comorbidity of heroin dependence: 11-year findings from the Australian Treatment Outcome Study. *Addiction*, 110(6), 986–993.
- Teesson, M., Marel, C., Darke, S., Ross, J., Slade, T., Burns, L., ... Mills, K. L. (2017). Trajectories of heroin use: 10-11-year findings from the Australian Treatment Outcome Study. *Addiction*.
- Teesson, M., Newton, N. C., & Barrett, E. L. (2012). Australian school-based prevention programs for alcohol and other drugs: A systematic review. *Drug and Alcohol Review*, 31(6), 731–736.
- Teesson, M., Slade, T., Swift, W., Mills, K., Memedovic, S., Mewton, L., ... Hall, W. (2012). Prevalence, correlates and comorbidity of DSM-IV cannabis use and cannabis use disorders in Australia. *Australian and New Zealand Journal of Psychiatry*, 46(12), 1182–1192.
- Theobald, H., Bygren, L. O., Carstensen, J., & Engfeldt, P. A. (2000). Moderate intake of wine is associated with reduced total mortality and reduced mortality from cardiovascular disease. *Journal of Studies on Alcohol*, 61, 652–656.
- Thomas, R. E., McLellan, J., & Perera, R. (2015). Effectiveness of school-based smoking prevention curricula: Systematic review and meta-analysis. *BMJ Open*, 5(3), e006976.
- Thompson, P. M., Hayashi, H. M., Simon, S. L., et al. (2004). Structural abnormalities in the brains of human subjects who use methamphetamine. *Journal of Neuroscience*, 24, 6028–6036.
- Tonigan, J. S., Miller, W. R., & Connors, G. J. (2000). Project MATCH client impressions about Alcoholics Anonymous: Measurement issues and relationship to treatment outcome. *Alcoholism Treatment Quarterly*, 18, 25–41.
- Tonstad, S., Tonnesen, P., Hajek, P., Williams, K. E., Billing, C. B., & Reeves, K. R. (2006). Varenicline Phase 3 Study Group. Effect of maintenance therapy with varenicline on smoking cessation: A randomized controlled trial. *JAMA*, 296, 64–71.
- Tran, G. Q., Haaga, D. A. F., & Chambless, D. L. (1997). Expecting that alcohol will reduce social anxiety moderates the relation between social anxiety and alcohol consumption. *Cognitive Therapy and Research*, 21, 535–553.
- True, W. R., Xiam, H., Scherrer, J. F., Madden, P., Bucholz, K. K., et al. (1999). Common genetic vulnerability for nicotine and alcohol dependence in men. *Archives of General Psychiatry*, 56, 655–662.
- Tsuang, M. T., Lyons, M. J., Meyer, J. M., Doyle, T., Eisen, S. A., et al. (1998). Co-occurrence of abuse of different drugs in men: The role of drug-specific and shared vulnerabilities. *Archives of General Psychiatry*, 55, 967–972.
- Vegting, Y., Reneman, L., & Booij, J. (2016). The effects of ecstasy on neurotransmitter systems: A review on the findings of molecular imaging studies. *Psychopharmacology*, 233(19–20), 3473–3501.
- Volkow, N. D., Swanson, J. M., Evins, A. E., DeLisi, L. E., Meier, M. H., Gonzalez, R., ... Baler, R. (2016). Effects of cannabis use on human behaviour, including cognition, motivation and psychosis: A review. *JAMA Psychiatry*, 73(3), 292–297.
- Volkow, N. D., Wang, G. J., Fischman, M. W., & Foltin, R. W. (1997). Relationship between subjective effects of cocaine and dopamine transporter occupancy. *Nature*, 386, 827–830.
- Volpicelli, J. R., Rhines, K. C., Rhines, J. S., Volpicelli, L. A., et al. (1997). Naltrexone and alcohol dependence: Role of subject compliance. *Archives of General Psychiatry*, 54, 737–743.
- Volpicelli, J. R., Watson, N. T., King, A. C., Sherman, C. E., & O'Brien, C. P. (1995). Effects of naltrexone on alcohol "high" in alcoholics. *American Journal of Psychiatry*, 152, 613–617.
- Wagenaar, A. C., Salois, M. J., & Komro, K. A. (2009). Effects of beverage alcohol price and tax levels on drinking: A meta-analysis of 1003 estimates from 112 studies. *Addiction*, 104(2), 179–190.
- Wagenaar, A. C., Tobler, A. L., & Komro, K. A. (2010). Effects of alcohol tax and price policies on morbidity and mortality: A systematic review. *American Journal of Public Health*, 100(11), 2270–2278.
- Wakefield, M. A., Coomber, K., Durkin, S. J., Scollo, M., Bayly, M., Spittal, M. J., ... Hill, D. (2014). Time series analysis of the impact of tobacco control policies on smoking prevalence among Australian adults, 2001–2011. *Bulletin of the World Health Organization*, 92(6), 413–422.
- Wakefield, M. A., Loken, B., & Hornik, R. C. (2010). Use of mass media campaigns to change health behaviour. *The Lancet*, 376(9748), 1261–1271.
- Walitzer, K. S., & Dearing, R. L. (2006). Gender differences in alcohol and substance use relapse. *Clinical Psychology Review*, 26, 128–148.

- Whelan, R., Watts, R. orr, C. A., Althoff, R. R., Artiges, E., Banaschewski, T., . . . Carvalho, F. M. (2014). Neuropsychosocial profiles of current and future adolescent alcohol misusers. *Nature*, 512(7513), 185–189.
- Wills, T. A., & Cleary, S. D. (1999). Peer and adolescent substance use among 6th–9th graders: Latent growth analysis of influence versus selection mechanisms. *Health Psychology*, 18, 453–463.
- Wills, T. A., Sandy, J. M., & Yaeger, A. M. (2002). Stress and smoking in adolescence: A test of directional hypotheses. *Health Psychology*, 21, 122–130.
- Wilsnack, R. W., Vogeltanz, N. D., & Wilsnack, S. C., et al. (2000). Gender differences in alcohol consumption and adverse drinking consequences: Cross-cultural patterns. *Addiction*, 95, 251–265.
- Woicik, P. A., Stewart, S. H., Pihl, R. O., & Conrod, P. J. (2009). The substance use risk profile scale: A scale measuring traits linked to reinforcement-specific substance use profiles. *Addictive Behaviours*, 34(12), 1042–1055.
- World Health Organization (WHO). (2011). *Global status report on alcohol and health*. Geneva: WHO.
- Yoast, R., Williams, M. A., Deitchman, S. D., & Champion, H. C. (2001). Report of the Council on Scientific Affairs: Methadone maintenance and needle-exchange programs to reduce the medical and public health consequences of drug abuse. *Journal of Addictive Diseases*, 20, 15–40.

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## ACKNOWLEDGEMENTS

Table 7.1: Australian Institute of Health and Welfare

Table 7.3: Australian Institute of Health and Welfare

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## CHAPTER 8

# Eating disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 8.1** describe the symptoms associated with anorexia nervosa, bulimia nervosa and binge-eating disorder and be able to distinguish between the different eating disorders
  - 8.2** describe the genetic, neurobiological, sociocultural and psychological factors implicated in the aetiology of eating disorders
  - 8.3** describe the treatments for eating disorders and the evidence supporting their effectiveness.
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## OPENING SCENARIO

Emily was 16 years old when her family GP referred her to an inpatient eating disorder program. Her mother had insisted on coming with her to the appointment with the GP as she had noticed Emily's dramatic weight loss over the past few weeks. The GP was most worried about Emily's low and irregular heart rate and that she had not had her period for the last 12 months. He immediately referred her to a child and adolescent hospital specialised in the treatment of eating disorders. Although Emily did not really think anything was wrong with her, the GP confronted her with the choice of admitting herself or being committed involuntarily.

As a child, Emily had been chubby and her mother had incessantly commented negatively on her appearance: 'A princess should be slim and tall, not like you!' Since then Emily had constantly worried about her weight and body shape. She started to restrict her daily food intake by only eating 'safe foods' (e.g., low calorie fruits) and setting strict eating rules. When she started to lose some weight due to her strict diet, she felt immensely proud and a sensation of control and mastery. She also loved the admiration from others: 'You've lost weight, right? You really look great!' However, she became increasingly afraid of gaining weight again and 'becoming fat'. She was especially concerned about her buttocks and abdomen, which she found far too large.

Emily continued to restrict her daily calorie intake even further and also started running excessively several times per week to lose more weight. Paradoxically, the more weight Emily lost, the more she felt under control but also that she needed to lose more weight. Daily, she would spend more and more time critically examining her body in the mirror and weighing herself. Even after her weight had dropped to dangerous levels, she still thought she was not thin enough.

When Emily's family and friends started to express concerns about her dramatic weight loss, she did not believe that her behaviour was problematic or dangerous. 'What's the problem with being thin and exercising?' she thought.

### QUESTIONS

1. Emily does not seem to realise that she has lost too much weight and has developed a dysfunctional eating style. Why is she not able to see this?
2. How could comments from Emily's mother and friends be related to her weight loss?

## Introduction

Many cultures are preoccupied with food. In Australia and New Zealand, but also in many Asian countries today, new restaurants abound and numerous magazines, blogs, websites and television shows are devoted to food and food preparation. There is an abundance of high caloric, palatable food, which is often cheaper than healthier choices. Perhaps not surprisingly, many people are overweight and obese: in Australia almost two-thirds of the adult population are overweight or obese (ABS, 2013). Dieting to lose weight is common, and the desire of many people, especially women, to be thinner has created a multibillion-dollar-a-year business. Given this intense interest in food and eating, it is not surprising that this aspect of human behaviour is subject to disorder.

Although clinical descriptions of eating disorders can be traced back many years, even centuries for anorexia nervosa, they appeared in the DSM for the first time in 1980 as one subcategory of disorders beginning in childhood or adolescence. Eating disorders became a distinct category in DSM-IV, reflecting the increased attention they had received from clinicians and researchers. In DSM-5, eating disorders are included in the chapter 'Feeding and eating disorders', which also includes childhood disorders such as pica (eating non-food substances for extended periods) and rumination disorders (repeated regurgitation of foods).

Like other mental illnesses, eating disorders are likely to be stigmatised. In a recent study, undergraduate students were presented with a vignette depicting a fictional woman with an eating disorder (anorexia nervosa) and were then asked to rate her on a number of dimensions (Griffiths, Mond, Murray, & Touyz, 2014).

Participants rated the woman depicted with an eating disorder as 'self-destructive and responsible for their conditions'. Male participants were particularly likely to believe that eating disorders were easy to overcome. In another study (Zwickert & Rieger, 2013), participants were randomly assigned to read a vignette about a woman with anorexia nervosa, an obese woman or a woman with skin cancer; participants also received differing information describing the woman as either blameworthy or non-blameworthy for her condition. Participants were most resistant to having social contact with the woman suffering from anorexia nervosa compared to the women with medical conditions, although attributing less blame to the woman with anorexia nervosa.

## 8.1 Clinical descriptions of eating disorders

**LEARNING OUTCOME 8.1** Describe the symptoms associated with anorexia nervosa, bulimia nervosa and binge-eating disorder and be able to distinguish between the different eating disorders.

We begin by describing anorexia nervosa and bulimia nervosa. The diagnoses of these two disorders share several clinical features. We then discuss binge-eating disorder, which is a new diagnostic category in the DSM-5.

### Anorexia nervosa

Emily, the adolescent girl described in the opening scenario, has **anorexia nervosa**. The term *anorexia* refers to loss of appetite and *nervosa* indicates that the loss is due to emotional reasons. The term is something of a misnomer because most people with anorexia nervosa actually do not lose their appetite or interest in food. On the contrary, while starving themselves, most people with the disorder become pre-occupied with food; they may read cookbooks constantly and prepare gourmet meals for their families.

Emily met all three features required for the diagnosis.

1. *Restriction of energy intake leading to significantly low body weight.* This usually means that the person weighs much less than is considered normal (e.g., having a **body mass index [BMI]** less than 18.5 for adults; see table 8.1) for that person's age and height. Weight loss is typically achieved through dieting, although purging (self-induced vomiting, or heavy use of laxatives or diuretics) and excessive exercise are also common.
2. *Intense fear of gaining weight and being fat or behaviour that interferes with gaining weight.* This fear is not reduced by weight loss. For those with anorexia nervosa there is no such thing as 'too thin'.
3. *Distorted body image or sense of body shape.* Even when emaciated, those with anorexia nervosa believe that they are overweight and that certain parts of their bodies, particularly the abdomen, hips and thighs, are too fat. To check on their body size, they typically weigh themselves frequently, measure the size of different parts of the body and gaze critically at their reflections in mirrors. Their self-esteem is closely linked to maintaining thinness.

Prior to the DSM-5, *amenorrhoea* (*loss of menstrual period*) was one of the diagnostic criteria for anorexia nervosa. It was removed, however, in DSM-5 because there are many reasons why women can stop having their menstrual period that do not have anything to do with weight loss. In addition, few differences have been found between women who have amenorrhoea and the other three features of anorexia nervosa, and those women who have the other three features but do not have amenorrhoea (Attia & Roberto, 2009).

#### DSM-5

##### DSM-5 criteria for anorexia nervosa

- People with anorexia nervosa undertake a restriction of food that leads to very low body weight; their body weight is significantly below normal.
- People with anorexia nervosa have an intense fear of weight gain or repeat behaviours that interfere with weight gain.
- People with anorexia nervosa experience body image disturbance.

**TABLE 8.1** Computing your body mass index (BMI)

WEIGHT lbs	100	105	110	115	120	125	130	135	140	145	150	155	160	165	170	175	180	185	190	195	200	205	210	215
kgs	45.5	47.7	50.0	52.3	54.5	56.8	59.1	61.4	63.6	65.9	68.2	70.5	72.7	75.0	77.3	79.5	81.8	84.1	86.4	88.6	90.9	93.2	95.5	97.7
HEIGHT in/cm	Underweight					Healthy						Overweight				Obese		Extremely obese						
5'0" - 152.4	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	37	38	39	40	41	42
5'1" - 154.9	18	19	20	21	22	23	24	25	26	27	28	29	30	31	32	33	34	35	36	36	37	38	39	40
5'2" - 157.4	18	19	20	21	22	22	23	24	25	26	27	28	29	30	31	32	33	33	34	35	36	37	38	39
5'3" - 160.0	17	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	32	32	33	34	35	36	37	38
5'4" - 162.5	17	18	18	19	20	21	22	23	24	24	25	26	27	28	29	30	31	31	32	33	34	35	36	37
5'5" - 165.1	16	17	18	19	20	20	21	22	23	24	25	25	26	27	28	29	30	30	31	32	33	34	35	35
5'6" - 167.6	16	17	17	18	19	20	21	21	22	23	24	25	25	26	27	28	29	29	30	31	32	33	34	34
5'7" - 170.1	15	16	17	18	18	19	20	21	22	22	23	24	25	25	26	27	28	29	29	30	31	32	33	33
5'8" - 172.7	15	16	16	17	18	19	19	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	32	32
5'9" - 175.2	14	15	16	17	17	18	19	20	20	21	22	22	23	24	25	25	26	27	28	28	29	30	31	31
5'10" - 177.8	14	15	15	16	17	18	18	19	20	20	21	22	23	23	24	25	25	26	27	28	28	29	30	30
5'11" - 180.3	14	14	15	16	16	17	18	18	19	20	21	21	22	23	23	24	25	25	26	27	28	28	29	30
6'0" - 182.8	13	14	14	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28	29
6'1" - 185.4	13	13	14	15	15	16	17	17	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27	28
6'2" - 187.9	12	13	14	14	15	16	16	17	18	18	19	19	20	21	21	22	23	23	24	25	25	26	27	27
6'3" - 190.5	12	13	13	14	15	15	16	16	17	18	18	19	20	20	21	21	22	23	23	24	25	25	26	26
6'4" - 193.0	12	12	13	14	14	15	15	16	17	17	18	18	19	20	20	21	22	22	23	23	24	25	25	26

As discussed in the chapter on diagnosis and assessment, DSM-5 added severity ratings for nearly all disorders. For anorexia nervosa, the severity ratings (see table 8.2) are based on BMI, which is consistent with the approach of the World Health Organization. BMI is calculated by dividing weight in kilograms by height in metres squared. A healthy weight BMI is between 18.5 and 25. To calculate your own BMI, see table 8.1. The BMI is not a perfect measure and BMI cut-offs may vary from country to country; some Asian countries (e.g., Japan, Singapore and Hong Kong) have introduced lower cut-offs for public health actions due to higher risks for cardiovascular diseases at substantial lower BMIs. However, BMI values lower than 18.5 are consistently used to indicate underweight. In addition, many people may have a higher or lower BMI for reasons that do not have to do with body fat. For example, someone who is very muscular will likely have a high BMI but will not be overweight or obese. By contrast, an elite runner may be very lean and have a low BMI but not have anorexia nervosa.

**TABLE 8.2** Severity ratings for anorexia nervosa in DSM-5

Rating	BMI range
Mild	≤ 17
Moderate	16–16.99
Severe	15–15.99
Extreme	< 15

The body image disturbance that accompanies anorexia nervosa has been assessed in several ways, most frequently by self-report questionnaires such as the Eating Disorders Inventory (Garner, Olmstead, & Polivy, 1983). The body dissatisfaction subscale of this questionnaire primarily asks about weight-related body sites; see table 8.3 for some items of the body dissatisfaction scale. In another type of assessment, people with anorexia nervosa are shown line drawings of women (or men) with varying body weights and asked to pick the one closest to their own and the one that represents their ideal shape (see figure 8.1). People with anorexia nervosa overestimate their own body size and choose a thin figure as their ideal. Despite this distortion in body size, people with anorexia nervosa are fairly accurate when reporting their actual weight (McCabe, McFarlane, Polivy, & Olmsted, 2001), perhaps because they weigh themselves frequently.

It is important to note that recent research shows key differences between female and male body disturbances: whereas females often report a strong ‘drive for thinness’ (see also table 8.3), males often want to be ‘bigger’, more muscular (Murray, Griffiths, & Mond, 2016).

DSM-5 continues to include two subtypes of anorexia nervosa, even though research calls into question the validity of these types. In the *restricting type*, weight loss is achieved by severely limiting food intake as illustrated in Emily’s case; in the *binge-eating/purging type* a person also regularly engages in binge eating and purging behaviour. Longitudinal research, however, suggests that the distinction between subtypes may not be all that useful (Eddy et al., 2002). Nearly two-thirds of women who initially met criteria for the restricting subtype had switched over to the binge-eating/purging type eight years later. A review of the subtype literature for the preparation of DSM-5 concluded that subtypes had limited predictive validity even though clinicians found them useful (Peat, Mitchell, Hoek, & Wonderlich, 2009). The fact that one of the top priorities for the DSM-5 was to be useful to clinicians may explain why these subtypes were retained despite their limited validity.

Anorexia nervosa typically begins in the early to middle teenage years, often after an episode of dieting and the occurrence of life stress. Community studies from Western countries report lifetime prevalence rates of 0.9 for anorexia nervosa among adult females (Hudson, Hiripi, Pope, & Kessler, 2007). However, an Australian study of female twins reported a higher lifetime prevalence of 1.9 percent for anorexia nervosa, which might be due to the comprehensive eating disorder assessments completed in this study. While the overall rate has been stable for several decades, there has been an increase in the high-risk group of 15- to 19-year-old girls. In short, the rates of anorexia nervosa in Western countries are not rising or falling (Smink, van Hoeken, & Hoek, 2012). However, the traditional picture that only adolescent Caucasian females in high-income Western countries are suffering from anorexia nervosa is no longer true. A review by Pike and Dunne (2015) evaluated the ‘Rise of eating disorders in Asia’ and concluded that increasing industrialisation and urbanisation in many Asian countries accompanied with a ‘Westernisation’ of (thin) beauty ideals has led to increasing rates of anorexia nervosa in some Asian countries (e.g., Hong Kong, Japan).

Despite being thin, people with anorexia nervosa believe that parts of their bodies are too fat and they spend a lot of time critically examining themselves in front of mirrors.



**TABLE 8.3** Subscales from the eating disorders inventory

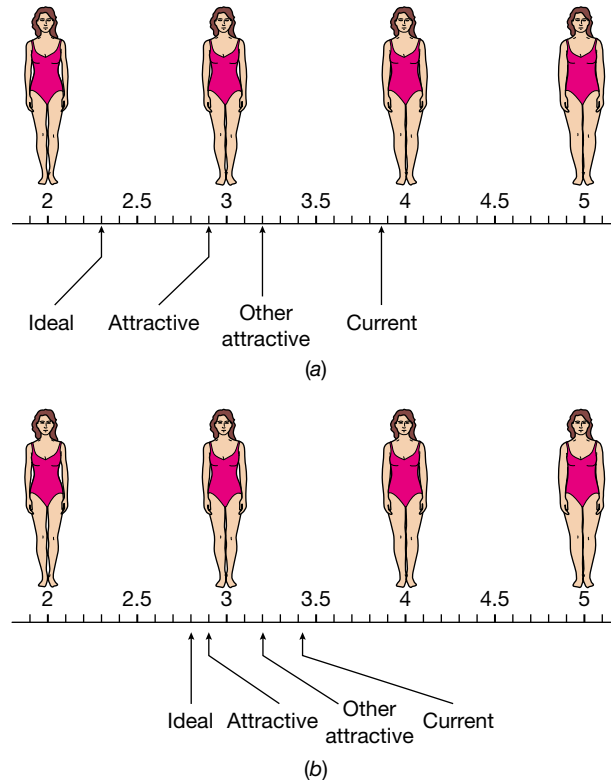
Drive for thinness	I think about dieting. I feel extremely guilty after overeating. I am preoccupied with the desire to be thinner.
Body dissatisfaction	I think that my thighs are too large. I think that my buttocks are too large. I think that my hips are too big.

**Note:** Responses use a six-point scale ranging from ‘always’ to ‘never’.

**Source:** Garner et al. (1983).

**FIGURE 8.1**

In this assessment of body image, respondents indicate their current shape, their ideal shape and the shape they think is most attractive to the opposite sex. The figure actually rated as most attractive by members of the opposite sex is shown in both panels. Ratings of women who scored high on a measure of distorted attitudes towards eating are shown in (a); ratings of women who scored low are shown in (b). The high scorers overestimated their current size and ideally would be very thin.



*Source:* Zellner, Harner, & Adler (1989).

Anorexia nervosa is at least 10 times more frequent in women than in men (Smink et al., 2012). When anorexia nervosa does occur in men, symptoms and other characteristics, such as personality ratings, are generally similar to those reported by women with the disorder (Strober et al., 2006). As we discuss more fully later, the gender difference in the prevalence of anorexia nervosa most likely reflects the greater cultural emphasis on women's bodies, which has promoted a thin shape as the ideal over the past several decades. Interestingly, a study comparing males with anorexia nervosa and muscle dysmorphia (a subtype of body dysmorphic disorder, with an obsessive preoccupation with being insufficiently muscular) found substantial overlap in symptoms between these two clinical groups (Murray et al., 2012), suggesting that some males with body image and eating concerns may be diagnosed with body dysmorphic disorder rather than with anorexia nervosa.

For both men and women, anorexia nervosa is frequently comorbid with depression, obsessive-compulsive disorder, specific phobias, panic disorder and various personality disorders (Cassin & von Ranson, 2005; Cederlöf et al., 2015; Fernandez-Aranda et al., 2007; Hughes, 2012; Swinbourne et al., 2012). Mortality rates for individuals with anorexia nervosa are high; suicide rates are also high with one in five individuals with anorexia nervosa who died committing suicide (Arcelus, Mitchell, Wales, & Nielsen, 2011).

### Physical consequences of anorexia nervosa

Self-starvation and use of laxatives to lose weight produce numerous undesirable biological consequences in people with anorexia nervosa. Blood pressure often falls, heart rate slows, kidney and gastrointestinal

problems develop, bone mass declines, the skin dries out, nails become brittle, hormone levels change and mild anaemia (i.e., decrease in red blood cells) may occur. Some people lose hair from the scalp and they may develop lanugo — a fine, soft hair — on their bodies as a means of keeping body temperature due to loss of fat tissue. Levels of electrolytes, such as potassium and sodium, are altered. These ionised salts, present in various bodily fluids, are essential to neural transmission and lowered levels can lead to tiredness, weakness, cardiac arrhythmias and even sudden death.

## Prognosis

Although approximately 50 percent of people with anorexia nervosa eventually recover, a substantial proportion will still experience some symptoms or remain chronically ill (Steinhausen, 2002). In addition, recovery often takes more than six years (Wade, Bergin, Tiggemann, Bulik, & Fairburn, 2006) and relapses are common before a stable pattern of eating and weight maintenance is achieved. As we discuss later, changing people's distorted views of themselves is very difficult, particularly in cultures that value thinness.

Anorexia nervosa is a life-threatening illness; death rates are 10 times higher among people with the disorder than among the general population and twice as high as among people with other psychological disorders. Mortality rates among women with anorexia nervosa range from 3 to 5 percent (Keel & Brown, 2010). Death most often results from physical complications of the illness — for example, congestive heart failure — and from suicide (Herzog et al., 2000). A recent longitudinal study found that death was more likely to occur among those who had anorexia nervosa the longest (ranging from 7 to 25 years) (Franko et al., 2013).

Anorexia nervosa can be a life-threatening condition. It is especially prevalent among young women who are under intense pressure to keep their weight low. Brazilian model Ana Carolina Reston died from the condition in 2006 at age 21.



## CLINICAL CASE

### Jing

Jing was the second child born to her parents. Both she and her brother became intensely involved in athletics at an early age, Jing in gymnastics and her brother in swimming. At age four, Jing was enrolled in gymnastics school, where she excelled. By the time she was nine, her mother had decided that Jing had outgrown the coaching abilities of the local instructors and began driving her to a nationally recognised coach several times a week. Over the next few years, Jing's trophy case swelled and her aspirations for a place on the Olympic team grew. As she reached puberty, though, her thin frame began to fill out, raising concerns about the effects of weight gain on her performance as a gymnast. She began to restrict her intake of food but found that after several days of semistarvation she would lose control and go on an eating binge. This pattern of dieting and binge eating lasted for several months and Jing's fear of becoming fat seemed to increase during that time. At age 13, she hit on the solution of self-induced vomiting. She quickly fell into a pattern of episodes of binge eating and vomiting three or four times per week. Although she maintained this pattern in secret for a while, eventually her parents caught on and initiated treatment for her.

### QUESTIONS

1. How did Jing end up in a vicious cycle of dieting and binge eating?
2. Were there some early warning signs Jing's parents or the coaches could have picked up on?

## Bulimia nervosa

Jing's behaviour in the clinical case illustrates the features of **bulimia nervosa**. *Bulimia* stems from a Greek word meaning 'ox hunger'. This disorder involves episodes of rapid consumption of a large amount of food, followed by compensatory behaviour, such as vomiting, fasting or excessive exercise, to prevent weight gain. The DSM defines *binge eating* as having two characteristics. First, it involves eating an excessive amount of food, that is, much more than most people would eat, within a short period of time (e.g., two hours). Second, it involves a feeling of losing control over eating (i.e., not being able to stop). Bulimia nervosa is not diagnosed if the binge eating and purging occur only in the context of anorexia nervosa and its extreme weight loss; the diagnosis in such a case is anorexia nervosa, binge-eating/purging type. The key difference between anorexia nervosa and bulimia nervosa is weight loss: people with anorexia nervosa lose a tremendous amount of weight, whereas people with bulimia nervosa do not.

### DSM-5

#### DSM-5 criteria for bulimia nervosa

- People with bulimia nervosa experience recurrent episodes of binge eating.
- People with bulimia nervosa experience recurrent compensatory behaviours to prevent weight gain, for example, vomiting.
- Body shape and weight are extremely important for self-evaluation.

In bulimia nervosa, binge eating typically occurs in secret; it may be triggered by stress and negative emotions, and it continues until the person is uncomfortably full (Smyth et al., 2007). In the case of Jing, she was likely to start binge eating after periods of stress associated with being an elite athlete. Foods that can be rapidly consumed, especially sweets such as ice-cream and cake, are usually part of a binge. One study found that women with bulimia nervosa were more likely to binge while alone and during the morning or afternoon. In addition, avoiding a craved food on one day was associated with a binge episode with that food the next morning (Waters, Hill, & Waller, 2001). Other studies show that binge eating is likely to occur after a negative social interaction — or at least the perception of a negative social exchange (Ranzenhofer et al., 2014).

Research suggests that although people with bulimia nervosa sometimes ingest enormous quantities of food during binge-eating episodes — often more than what a person eats in an entire day — there is wide variation in the caloric content consumed during these episodes (Wolfe, Baker, Smith, & Kelly-Weeder, 2009). People report that they lose control during a binge, even to the point of experiencing something akin to what happens in addiction (Smith & Robbins, 2013), perhaps losing awareness of their behaviour. They are usually ashamed of their behaviour and try to conceal it.

After the binge-eating episode, feelings of discomfort, disgust and fear of weight gain lead to the second step of bulimia nervosa — the inappropriate compensatory attempt to undo the caloric effects of the binge eating. People with bulimia nervosa most often induce vomiting, take laxatives or diuretics (these behaviours are known as purging), fast or exercise excessively in order to prevent weight gain.

Although many people binge occasionally and some people also purge, the DSM-5 diagnosis of bulimia nervosa requires that the episodes of binge eating and compensatory behaviour occur at least once a week for three months. Is once a week a well-established cut-off point? Probably not. The frequency changed from twice a week in DSM-IV-TR to once a week in DSM-5 because few differences were found between people with binge-eating episodes twice a week and those who do so less frequently (Wilson & Sysko, 2009).

Similarly to those with anorexia nervosa, the self-esteem of people with bulimia nervosa depends heavily on maintaining a specific weight. Whereas people without eating disorders typically under-report their weight and say they are taller than they actually are, people with bulimia nervosa are more accurate in their report (McCabe et al., 2001). Yet, people with bulimia nervosa are also likely to be highly dissatisfied with their bodies.

DSM-IV-TR included subtypes of bulimia nervosa, but because the subtypes did not have much validity or clinical utility, they were removed from DSM-5. New severity ratings in the DSM-5 are based on the number of compensatory behaviours in a week (see table 8.4).

**TABLE 8.4** Severity ratings for bulimia nervosa in DSM-5

Rating	Number of compensatory behaviours/week
Mild	1–3
Moderate	4–7
Severe	8–13
Extreme	14 or more

Bulimia nervosa typically begins in late adolescence or early adulthood. About 90 percent of people with bulimia nervosa are women and according to recent evidence the prevalence among women is thought to be about 1 to 2 percent of the population (Smink et al., 2012). Many people with bulimia nervosa were somewhat overweight before the onset of the disorder and the binge eating often started during an episode of dieting. Although both anorexia nervosa and bulimia nervosa among women typically begin in adolescence, they can persist into adulthood and middle age (Hill, Haslett, & Kumar, 2001; Slevec & Tiggemann, 2011).

Bulimia nervosa is comorbid with numerous other diagnoses, notably depression, anxiety disorders, substance use, conduct disorder and personality disorders (Hudson et al., 2007; Wade, Bulik, Neale, & Kendler, 2000). Suicide rates are higher among people with bulimia nervosa than in the general population (Crow et al., 2009), but substantially lower among people with anorexia nervosa (Smink et al., 2012).

Which comes first, bulimia nervosa or the comorbid disorders? A prospective eight-year study examined the relationship between bulimia nervosa and depression symptoms among adolescent girls (Presnell, Stice, Seidel, & Madeley, 2009) and found that symptoms of bulimia nervosa predicted the onset of depression symptoms. However, the converse was also true: depression symptoms predicted the onset of bulimia nervosa symptoms. Thus, it appears each disorder increases the risk for the other.

### Physical consequences of bulimia nervosa

Bulimia nervosa is a serious disorder with many unfortunate medical consequences (Mehler, Krantz, & Sachs, 2015). For example, frequent purging can cause potassium depletion. Heavy use of laxatives induces diarrhoea, which can also lead to changes in electrolytes and cause irregularities in the heart-beat. Recurrent vomiting has been linked to menstrual problems and may lead to tearing of tissue in the stomach and throat, and to loss of dental enamel as stomach acids eat away at the teeth, which become ragged. The salivary glands may become swollen.

Although less common than in anorexia nervosa, menstrual irregularities, including amenorrhoea, can occur, even though people with bulimia nervosa typically have a normal BMI (Gendall, Bulik, Joyce, McIntosh, & Carter, 2000). Mortality rates for bulimia nervosa seem to be lower than for anorexia nervosa (Arcelus et al., 2011), but one study of nearly 1000 women with bulimia nervosa found the mortality rate to be nearly 4 percent (Crow et al., 2009).

### Prognosis

Long-term follow-ups of people with bulimia nervosa reveal that around 45 percent fully recover, although nearly one-quarter remain symptomatic and have a chronic course (Steinhausen & Weber, 2009).

#### CLINICAL CASE

##### Amy

Amy, a 27-year-old woman from a small rural town in New Zealand, described a lifelong struggle with her weight. She was described as 'chubby' as a child and peers often called her 'fatty'. She went on several diets when she was young, but none of them were successful. Currently, Amy is obese with a BMI of 35.

Amy had experienced several episodes of binge eating beginning at age 18, when she first left home for university. After being left out of a social group on campus, she retreated to her dorm room alone, where she ate two large pizzas and two bags of crisps. After the binge-eating episode, she felt very full and went to sleep. Amy found herself doing this as often as twice a week throughout university. She was not always hungry when she engaged in binge eating, but even though she felt extremely full, she could not stop eating. Afterwards, she felt ashamed and angry with herself for having eaten so much. She gained 30 kilograms during her university years.

Amy reported that she currently has binge-eating episodes at least once a week, typically when she has had a very stressful day at work. She has recently confided in a friend about her troubled eating and her friend recommended that she seek treatment at the local mental health clinic.

### QUESTIONS

1. What was the preceding event of Amy's first binge-eating episode and how did she feel before and after it?
2. How does Amy's BMI compare to other people suffering from binge-eating disorder?

## Binge-eating disorder

**Binge-eating disorder** was first included as a diagnosis in DSM-5 (it was considered a diagnosis in need of further study in DSM-IV-TR). This disorder includes recurrent binge-eating episodes (once per week for at least three months), with a lack of control during the episode and a feeling of distress about binge eating, as well as other characteristics, such as rapid eating and eating alone. It is distinguished from anorexia nervosa by the absence of weight loss and from bulimia nervosa by the absence of compensatory behaviours (i.e., purging, fasting or excessive exercise). Often, people with binge-eating disorder are **obese** (i.e., have a BMI greater than 30). With the current explosion in the prevalence of obesity, it is perhaps not surprising that research on binge-eating disorder continues to increase (Attia et al., 2013). It is important to point out, however, that not all obese people meet criteria for binge-eating disorder. Recent research suggests that anywhere from 5 to 30 percent of obese people meet the criteria for binge-eating disorders (Brownley et al., 2016). Obese individuals who engage in regular binge eating report lower quality of life (Rieger, Wilfley, Stein, Marino, & Crow, 2005). For further discussion of obesity, see focus on discovery 8.1.

### FOCUS ON DISCOVERY 8.1

#### Obesity: a twenty-first century epidemic?

Obesity is not an eating disorder, although it is a serious public health problem. It is estimated that the total direct cost for obesity and being overweight in 2005 in Australia was \$21 billion. The direct costs of health conditions usually include both health costs (e.g., hospitalisation, prescription medication) and non-health costs (e.g., transport to hospitals, supported accommodation). In addition, obesity and being overweight are linked to other health care costs, such as lost productivity through workplace absence or early retirement, but also premature death. In the Australian health care system these indirect costs (e.g., disability pension, mobility allowance) result in additional costs of \$35.6 billion per year; adding to a total annual cost of \$56.6 billion for obesity and being overweight (Colagiuri et al., 2010). The cost burden of obesity and being overweight are also considerable in other countries in the Asia-Pacific region. A study from New Zealand reports that health care costs associated with obesity and being overweight were estimated to be more than 4 percent of New Zealand's total health care expenditure in 2006 (Lal, Moodie, Ashton, Siahpash, & Swinburn, 2012). Cost analyses from Asian countries including China (Zhao et al., 2008), South Korea (Kang, Jeong, Cho, Song, & Kim, 2011) and Thailand (Pitayatiananan et al., 2014) confirm that increasing rates of overweight and obesity add considerable costs to national health care budgets.

Why are the health care costs so high? Obesity is linked to many health problems, including diabetes, hypertension, cardiovascular disease and several forms of cancer. Studies have found that the risks of coronary heart disease and type 2 diabetes increase steadily with increasing BMI (e.g., Joshy et al., 2014). According to a report of the Australian Institute of Health and Welfare (AIHW), a high BMI was considered the second strongest risk factor for the total global burden of disease in 2010 in Australia and New Zealand (AIHW, 2014); that means that a high BMI accounted for about 9 percent of the total impact on ill-health and premature death. A large population-based study assessing globally the risk for one lost year of 'healthy life' in 2013 estimated that the leading risk in Australia and New Zealand was a high BMI (Forouzanfar et al., 2015).

Globally, the proportion of overweight and obese people is increasing at an alarming rate. A large study across 188 countries from 1980 to 2013 found that nearly one-third of the world's population (approximately 2.1 billion people) were overweight or obese in 2013, a 10 percent increase over the last 33 years (The GBD 2013 Obesity Collaboration et al., 2014). According to the study, New Zealand and Australia have the highest rates of adult and child obesity in the region. Australia reports an alarming 14 percent increase in adult rates of being overweight over the last three decades. Data from the 2014–15 National Health Survey show that being overweight or obese is rather the norm: almost two in three Australian adults are overweight or obese (almost 28 percent of the adult population are obese; that is, 4.9 million people) and more than one in four children aged 5–17 years are overweight or obese (ABS, 2015).

There is a global trend that overweight and obesity in children and adolescents is on the rise, both in developed and developing countries (The GBD 2013 Obesity Collaboration et al., 2014). However, there has been some positive news: although childhood obesity levels in Australia remain high, the increase has paused, with almost no change over the last decade.

Although obesity rates are still considerably lower in the Asia-Pacific region (Malaysia has the highest obesity prevalence of 14 percent), compared with Australia and New Zealand, recently there has been an alarming rise in that region; for example, obesity rates have increased in Malaysia by around 5 percent in the last 10 years (The GBD 2013 Obesity Collaboration et al., 2014). Furthermore, Asian populations are thought to have an increased risk for type 2 diabetes and cardiovascular diseases with substantially lower BMIs than the existing cut-offs for being overweight (Boffetta et al., 2011). Thus, the World Health Organization has revised BMI risk categories for Asian populations and recommends a BMI of 23 or higher as the trigger point for public health action (WHO Expert Consultation, 2004).

Obesity is also a serious health problem among Indigenous communities. Aboriginal and Torres Strait Islander Australians are 1.6 times as likely to be obese as non-Indigenous Australians; two-thirds (66 percent) of Indigenous Australians aged 15 years and older are currently overweight or obese (ABS, 2013). In addition, the lifetime risk for developing type 2 diabetes is greater for Aboriginal Australians at any BMI, with obesity being a potent predictor of diabetes incidence (Wang, Hoy, & Si, 2010).

Why are so many people overweight? A number of factors play a role, including the environment we live in. In the book *Food Fight* by Brownell and Horgen (2004), the authors describe our 'toxic', obesogenic environment with respect to little physical activity, sedentary lifestyles and the availability of bad food options. Food is available at any time, but often it is easier and cheaper to go with the unhealthy but highly palatable option. Another recently published book by Cohen (2014) entitled *A Big Fat Crisis* reports that the availability and amount of all food, not just fast food, has increased exponentially in the past decades. The author estimates that people in the United States pay far less for food now than they once did, spending less than 10 percent of their income on food compared to 25 percent in the 1920s (Cohen, 2014).

Nevertheless, food purchases are influenced by actual food costs, especially for lower socioeconomic incomes. Precariously, a study from Australia found that non-essential, energy-dense and nutrient-poor food groups (e.g., ice-cream, cakes and biscuits, soft drinks) are becoming increasingly cheaper than essential nutritious food groups (e.g., milk, bread, cereals, meat, fish, fruit, vegetables, eggs). Indeed, there seems to be an association between social factors (e.g., low socioeconomic status) and the prevalence of obesity. For example, a study from the metropolitan area of Melbourne found that residents living in the lowest-income neighbourhoods had 2.5 times more exposure to fast-food outlets compared to residents living in areas with the highest incomes (Reidpath, Burns, Garrard, Mahoney, & Townsend, 2002).

People eat in restaurants more than ever before, calorie counts in cookbooks are higher and portion sizes of foods, both in restaurants and in grocery stores, are larger. In fact, most people do not know the portion size of most foods recommended by the Australian Dietary Guidelines (National Health and Medical Research Council, 2013). A 375 mL can of soft drink is not one serving, but rather between two and two and a half servings. Australians eat a daily average of 10 380 kJ (2480 calories) for men or 7080 kJ (1691 calories) for women, representing a significant increase of energy intake (approximately 350 kJ or 84 calories) over the time period studied — 1983 to 1995 (Cook, Rutishauser, & Seelig, 2001). Ever-increasing portion sizes, as well as the greater availability of unhealthy foods, affect the amount we eat. Even our cookbooks can influence what we eat. In an analysis of 18 recipes from *The Joy of Cooking* — one of the most published cookbooks in the United States since 1936 — Wansink and Payne (2009) found that calories in these recipes increased by a third between 1936 and 2006.

We are all subject to the continuing impact of advertisements, especially those promoting alluring high-calorie products such as snack foods, desserts and meals at fast-food restaurants. For example, the advertising budget for Coke and Pepsi combined was \$3 billion in 2001 (Brownell & Horgen, 2004). The Australian food industry spent more than \$400 million on marketing in 2010. Many dollars of the food industries' advertising budget is spent on marketing junk food to the most vulnerable, our children. Following recommendations from the Australian National Preventive Health Taskforce (Obesity Working Group Australia, 2009) to limit children's exposure to unhealthy foods, in 2009 representatives of the food industry signed an initiative not to advertise food products to children unless representing healthy dietary choices. However, a systematic review of studies examining food advertising on Australian television since 2009 concludes that many food companies continue to advertise unhealthy foods at times when children watch television (Smithers, Lynch, & Merlin, 2014). A task force of the American Psychological Association concluded in 2004 that television advertisements of unhealthy foods (e.g., sugary cereals, soft drinks) contribute to unhealthy eating habits of children under eight years of age, largely because these children lack the requisite cognitive skills to discern truth from advertising (Kunkel et al., 2004). Showing the direct effects of TV marketing, one study provided snacks to children as they watched a TV cartoon containing commercials for snack foods or for other products. Children randomly assigned to watch the show with the snack food commercials ate more snacks than children assigned to watch the cartoon with non-food advertising (Harris, Bargh, & Brownell, 2009). Along with increasing energy intake and bad food choices, many people, including children, have become more sedentary, spending more time working or playing on computers or smartphones and watching TV than ever before. Australians typically spend between 50–70 percent of their waking hours being sedentary (usually sitting) (Healy et al., 2008).

In addition to environmental factors, heredity plays a role in obesity. It has been estimated that between 25 and 40 percent of the variance in obesity is attributed to behavioural genetic factors (Brownell & Horgen, 2004). Recent evidence, however, suggests that for children up to the age of 18 years, genetic factors might play an even stronger role than environmental factors (Silventoinen, Rokholm, Kaprio, & Sorensen, 2010). These factors, however, only have an impact when they interact with environmental factors. Heredity could produce its effects by regulating metabolic rate, impacting the hypothalamus or influencing the production of enzymes that make it easier to store fat and gain weight. Molecular genetics studies have identified numerous possible genes, gene regions or variations of single nucleotides (polymorphism) that might contribute to obesity. Genome-wide association studies (GWAS) have identified more than 52 gene locations (*loci*) associated with obesity-related traits (Albuquerque, Stice, Rodríguez-López, Manco, & Nóbrega, 2015). Although genetic factors tell part of the story, they do not tell the whole of it. Clearly, the environment plays a critical role. For example, a recent study of over 500 000 women found that mothers' weight gain during pregnancy was more important in predicting high birth weight among babies than genetic factors (Ludwig & Currie, 2010).

Furthermore, stress and its associated negative moods can induce eating in some people (e.g., Kenardy, Arnow, & Agras, 1996). For example, Chua, Touyz and Hill (2004) experimentally manipulated the mood of 40 obese females with binge-eating disorder by letting them watch either a sad or a neutral 20-minute film excerpt. Directly after watching the film, plates of chocolate were offered to all participants. Results revealed that participants watching the sad film reported lower mood and ate significantly more chocolate. Research also shows that negative mood usually gets worse after eating, or at least after binge eating (Haedt-Matt & Keel, 2011). In addition, trying to suppress negative feelings is not effective in avoiding the tendency to eat when feeling bad. In one study, people who were asked to suppress their feelings of sadness after watching a sad film ate more ice-cream than those who were asked to just watch the film (Vohs & Heatherton, 2000).

The stigma associated with being overweight or obese is a serious issue. The Australian version of *The Biggest Loser*, a TV 'reality' show devoted to watching obese people struggle to lose weight, regularly reaches a large audience. The show, where participants are subjected to a tough exercise and eating regimen, is primetime entertainment, but certainly not the most effective way to reduce stigma. Criticism asserts that such a format increases negative stereotypes about overweight or obese people, humiliates participants publicly and sets unrealistic expectations about weight loss.

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Obesity is very prevalent, particularly in Australia and New Zealand.



Stigma can also perpetuate the idea that obesity is simply a matter of personal responsibility — the belief that if people would just eat less and exercise more, obesity would not be a problem. However, we have seen that this is only a small part of the story of why people are overweight. Given the multitude of factors contributing to obesity just noted, such a simple solution is not reasonable. We make hundreds of food-related decisions every day, but are only aware of a tiny fraction of them (Wansink & Sobal, 2007). Personal responsibility is important and people can and should make better choices about what and how much they eat. Nevertheless, other environmental factors, such as availability, cost and transportation, can sometimes work against such choices. Furthermore, our automatic attentional and cognitive process also impact on such decision making.

What can governments or policy makers do to prevent further population weight gain? Policy strategies to increase healthy eating and physical activity among communities have been available in the literature to governments for many years (e.g., Sacks, Swinburn, & Lawrence, 2008). In the past several years, Australia has passed a number of legislative and policy changes initiated to try to curb obesity. In 2008, the Australian Federal Government announced a major investment of more than \$900 million to reduce lifestyle-related chronic diseases (for many, obesity is a strong risk factor) and funded several community-based obesity prevention initiatives. As one of these initiatives, in 2013 the Australian National Preventive Health Agency (ANPHA) launched *Shape up Australia*, a program providing information on healthy weight, diet and physical activity. However, the preventive health funding was ceased in the federal Australian budget of 2014. This has raised criticism from those who argue for political priority in obesity prevention (Moodie, Tolhurst, & Martin, 2016). Further research is required to understand the impact and effectiveness of such preventative programs. Clear and understandable food labelling is another field where policy makers can help consumers with legislative action to make

healthier food choices. Allowing people to satisfy their healthy food preferences by labelling unhealthy options clearly is one of the preventive recommendations made by a global network of public-interest organisations and researchers (Swinburn et al., 2015). Food industry behaviour is slowly changing, in selected instances. Under pressure from consumers, public officials and health advocates, three big soft drink producers pledged to reduce calories in sugary drinks by 20 percent before 2025. Whether such an effort will help reduce global obesity rates remains to be seen. Still, much work needs to be done to help halt the obesity epidemic.

### QUESTIONS

1. As described in the book *Food Fight*, what is a 'toxic', obesogenic environment and how could it lead to obesity?
2. How is stress related to overeating?

Binge-eating disorder was added to DSM-5 based on a growing body of research supporting its validity as a diagnostic category (Attia et al., 2013; Wonderlich, Gordon, Mitchell, Crosby, & Engel, 2009). Severity ratings for this disorder are shown in table 8.5 and are based on the number of binge-eating episodes per week. Many people with binge-eating disorder have a history of dieting; however, there is a subgroup of individuals suffering from binge-eating disorder who report that binge eating preceded their first diet (Grilo & Masheb, 2000). Binge-eating disorder is comorbid with several disorders, including mood disorders, anxiety disorders, ADHD, conduct disorder and substance use disorders (Kessler et al., 2013; Wonderlich et al., 2009). Risk factors for developing binge-eating disorder include childhood obesity, critical comments about being overweight, weight-loss attempts in childhood, low self-concept, depression, and childhood physical or sexual abuse (Fairburn et al., 1998; Striegel-Moore et al., 2005).

**TABLE 8.5** Severity ratings for binge-eating disorder in DSM-5

Rating	Number of binge-eating episodes/week
Mild	1–3
Moderate	4–7
Severe	8–13
Extreme	14 or more

Binge-eating disorder is more prevalent than either anorexia nervosa or bulimia nervosa (Kessler et al., 2013; Wade et al., 2006). In a recent study of several countries, the lifetime prevalence ranged from 0.2 to 4.7 percent (Kessler et al., 2013); in an Australian study with female twins, 2.9 percent of women met criteria for binge-eating disorder (Wade et al., 2006). Binge-eating disorder is more common in women than men, although the gender difference is not as great as it is in anorexia nervosa or bulimia nervosa (Kessler et al., 2013).

### DSM-5

#### DSM-5 criteria for binge-eating disorder

- People with binge-eating disorder experience recurrent binge-eating episodes.
- Binge-eating episodes include at least three of the following:
  - eating more quickly than usual
  - eating until overly full
  - eating large amounts even if not hungry
  - eating alone due to embarrassment about large food quantity
  - feeling bad (e.g., disgusted, guilty or depressed) after the binge-eating episode.
- No compensatory behaviour is present.

## Physical consequences of binge-eating disorder

Similarly to anorexia nervosa and bulimia nervosa, binge-eating disorder has physical consequences. Many of them are likely a function of associated obesity, including increased risk of type 2 diabetes, cardiovascular problems, chronic back pain and headaches, even after controlling for the independent effects of other comorbid disorders (Kessler et al., 2013). Other research shows that a number of physical problems are present among people with binge-eating disorder that are independent from co-occurring obesity, including sleep problems, anxiety, depression, irritable bowel syndrome and, for women, early onset of menstruation (Bulik & Reichborn-Kjennerud, 2003).

## Prognosis

Perhaps because it is a relatively new diagnosis, fewer studies have assessed the prognosis of binge-eating disorder. Research so far suggests that between 25 and 82 percent of people recover (Keel & Brown, 2010). One retrospective study found that people reported experiencing binge-eating disorder on average for more than 14 years, which is much longer than for people with anorexia nervosa or bulimia nervosa (Pope et al., 2006). However, another recent epidemiological study of binge-eating disorder in several countries reported an average duration of just over four years (Kessler et al., 2013).

## 8.2 Aetiology of eating disorders

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**LEARNING OUTCOME 8.2** Describe the genetic, neurobiological, sociocultural and psychological factors implicated in the aetiology of eating disorders.

As with other mental disorders, a single factor is unlikely to cause an eating disorder. Several areas of current research — genetics, neurobiology, sociocultural pressures to be thin, personality, the role of the family and the role of environmental stress — suggest that eating disorders result when several influences converge in a person's life.

### Genetic factors

Eating disorders run in families. First-degree relatives of women with anorexia nervosa are more than 10 times more likely than average to have the disorder themselves (Strober, Freeman, Lampert, Diamond, & Kaye, 2000). Similar results are found for bulimia nervosa, where first-degree relatives of women with bulimia nervosa are about four times more likely than average to have the disorder (Strober et al., 2000). Furthermore, first-degree relatives of women with eating disorders appear to be at higher risk for anorexia nervosa or bulimia nervosa (Strober et al., 2000). Although eating disorders are quite rare among men, one study found that first-degree relatives of men with anorexia nervosa were at greater risk for having anorexia nervosa (not bulimia nervosa) than relatives of men without anorexia nervosa (Strober, Freeman, Lampert, Diamond, & Kaye, 2001). One behaviour genetics study (Hudson et al., 2006) found that relatives of obese people with binge-eating disorder were more than twice as likely to have binge-eating disorder themselves (20 percent) than were relatives of obese people without binge-eating disorder (less than 10 percent).

Twin studies of eating disorders also suggest a genetic influence. Most studies of both anorexia nervosa and bulimia nervosa report higher monozygotic (MZ) than dizygotic (DZ) concordance rates (Bulik, Sullivan, Wade, & Kendler, 2000) and show that genes account for a portion of the variance among twins with eating disorders (Wade et al., 2000). On the other hand, research has shown that non-shared/unique environmental factors (see chapter 1), such as different interactions with parents or different peer groups, also contribute to the development of eating disorders (Klump, Wonderlich, Lehoux, Lilienfeld, & Bulik, 2002). For example, in a study of 7000 female twins, 62 percent of the variance in symptoms of bulimia nervosa was attributable to genetic factors compared to 38 percent attributable to unique environmental factors (Bulik et al., 2010). Research also suggests that key features of eating disorders,

such as dissatisfaction with one's body or a strong desire to be thin, binge eating and preoccupation with weight, are also heritable (Wade, Treloar, Heath, & Martin, 2009). The largest GWAS study to date, which included nearly 3000 people with anorexia nervosa, identified a number of single nucleotide polymorphisms (SNPs; see chapter 1) that were associated with anorexia nervosa (Boraska, Franklin, Floyd et al., 2014). Even though this sample size sounds large, it was not large enough for the findings to hold up during replication, something that GWAS researchers frequently do to avoid reporting findings that could be due to chance. The results of all the genetic studies are consistent with the possibility that genes play a role in eating disorders, but studies showing how genetic factors interact with the environment need further attention (Yilmaz, Hardaway, & Bulik, 2015).

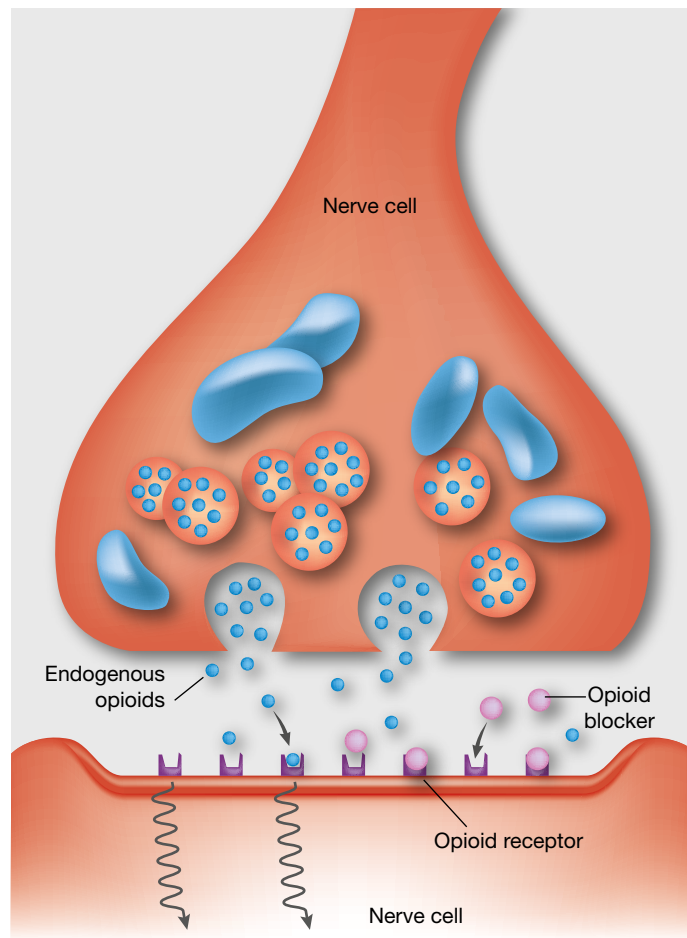
## Neurobiological factors

The hypothalamus is a key brain centre for regulating hunger and eating; thus, it is not surprising that the hypothalamus has been proposed to play a role in anorexia nervosa. The level of some hormones regulated by the hypothalamus, such as cortisol, is indeed different in people with anorexia nervosa. However, rather than causing the disorder, these hormonal differences occur as a result of self-starvation and levels return to normal after weight gain (Støving, Hangaard, Hansen-Nord, & Hagen, 1999).

Also, some endogenous opioids (e.g., beta-endorphin) have been hypothesised to play a role in anorexia nervosa, bulimia nervosa and binge-eating disorder. Endogenous opioids are substances produced by the body that reduce pain sensations, enhance mood and suppress appetite, and are also released during starvation. Starvation among people with anorexia nervosa may increase the levels of endogenous opioids, resulting in a positively reinforced mood state (Marrazzi et al., 1990). Furthermore, the excessive exercise seen among some people with eating disorders increase opioids and might thus be reinforcing. In contrast to anorexia nervosa, studies of people with bulimia nervosa found low levels of the endogenous opioid beta-endorphin (Bailer & Kaye, 2003) (see figure 8.2). However, it is important to note that these findings demonstrate that, while low levels of opioids are seen concurrently with bulimia nervosa, such levels are not necessarily seen before the onset of the disorder. In other words, we cannot conclude that low levels of opioids are a cause of bulimia nervosa or an effect of changes in food intake or purging. The same is true for binge-eating disorder. Animal studies have shown that binge eating leads to changes in the opioid system (Bello, Patinkin, & Moran, 2011) but not that changes in the opioid system lead to more binge eating.

Finally, some research has focused on two neurotransmitters (Kaye, 2008): serotonin, which is related to eating and satiety (feeling full), and dopamine, which is related to the rewarding/pleasing aspects of food. Animal research has shown that serotonin promotes satiety. Therefore, the binge-eating episodes in people with bulimia nervosa or binge-eating disorder could result from a serotonin deficit that causes them not to feel full after eating. Animal research has also shown that food restriction interferes with serotonin synthesis in the brain. Thus, among people with anorexia nervosa, the severe food intake restrictions could interfere with the serotonin system. Researchers have examined levels of serotonin metabolites among people with anorexia nervosa and bulimia nervosa. Several studies have found low levels of serotonin metabolites among people with anorexia nervosa (Kaye, Frank, Bailer, & Henry, 2005) and bulimia nervosa (Bailer & Kaye, 2011). Lower levels of a neurotransmitter's metabolites are one indicator that the neurotransmitter activity is underactive. In addition, people who have recovered from anorexia nervosa continue to have lower serotonin activity, suggesting that this neurotransmitter imbalance may be a trait-related disturbance rather than a symptom of malnutrition (Frank et al., 2002). Similar results were found for individuals who had recovered from bulimia nervosa (Kaye et al., 2001). The antidepressant drugs that can be effective treatments for some people with anorexia nervosa and bulimia nervosa (discussed later) are known to increase serotonin activity, adding to the possible importance of serotonin. Serotonin, though, is more likely linked to the comorbid depression often found in anorexia nervosa and bulimia nervosa.

**FIGURE 8.2** Endogenous opioid systems in the brain



Researchers have also examined the role of dopamine in eating behaviour. Studies with animals have shown that dopamine is linked to the pleasurable aspects of food that compel an animal to seek food (e.g., Berridge & Robinson, 1998). Brain-imaging studies in humans have shown how dopamine is linked to the motivation to obtain food and other pleasurable or rewarding things. In one study with healthy people, participants were presented with smells and tastes of food while undergoing a PET scan (Volkow et al., 2002). The participants also filled out a measure of dietary restraint (see table 8.6). People who scored higher on dietary restraint exhibited greater dopamine activity in the dorsal striatum area of the brain during the presentation of food. This finding suggests that people who restrain their eating may be more sensitive to food cues, since one of the functions of dopamine is to signal the salience of particular stimuli. Whether or not these findings will be relevant to people with eating disorders remains to be seen.

A small fMRI study of 14 women with anorexia nervosa and 14 women without an eating disorder found that women with anorexia nervosa reported feeling more positively about pictures of underweight women compared to pictures of normal or overweight women (Fladung et al., 2010). The women without anorexia nervosa felt more positively when viewing pictures of normal-weight women. Brain activation matched these ratings of feelings: women with anorexia nervosa showed greater activation in the ventral striatum, an area of the brain linked to dopamine and reward (see also the chapter on mood disorders), than women without anorexia nervosa when viewing pictures of underweight women.

**TABLE 8.6    The restraint scale**

1. How often are you dieting? Never; rarely; sometimes; often; always.
2. What is the maximum amount of weight (in kilograms) you have ever lost within one month? 0–2.5; 2.5–5; 5–7.5; 7.5–10; 10+.
3. What is your maximum weight gain (in kilograms) within a week? 0–0.5; 0.5–1; 1–1.5; 1.5–2.5; 2.5+.
4. In a typical week, how much does your weight fluctuate? 0–0.5; 0.5–1; 1–1.5; 1.5–2.5; 2.5+.
5. Would a weight fluctuation of 2.5 kilograms affect the way you live your life? Not at all; slightly; moderately; very much.
6. Do you eat sensibly in front of others and splurge alone? Never; rarely; often; always.
7. Do you give too much time and thought to food? Never; rarely; often; always.
8. Do you have feelings of guilt after overeating? Never; rarely; often; always.
9. How conscious are you of what you are eating? Not at all; slightly; moderately; extremely.
10. How many kilograms over your desired weight were you at your maximum weight? 0–0.5; 0.5–3; 3–5; 5–10; 10+.

*Source:* Adapted from Polivy, Herman, & Howard (1988).

Another study found that women with either anorexia nervosa or bulimia nervosa had greater expression of the dopamine transporter gene *DAT* (Frieling et al., 2010). Recall from chapter 1 that a gene is ‘turned on’, or expressed, as it interacts with different aspects of the environment. The expression of *DAT* influences the release of a protein that regulates the reuptake of dopamine back into the synapse. This study also found that women with either eating disorder exhibited less expression of another dopamine gene called *DRD<sub>2</sub>*. Other studies have found disturbances in the *DRD<sub>2</sub>* gene only among women with anorexia nervosa (Bergen et al., 2005). These findings point to the role of dopamine in eating disorders and will need to be replicated in future studies. Interestingly, animal studies of binge eating indicate that binge eating on sucrose (sugar) leads to increased release of dopamine in the striatum and it has different effects on two dopamine receptors: more binding of *D<sub>1</sub>* receptors in the ventral (bottom) part of the striatum but less binding of *D<sub>2</sub>* receptors in the dorsal (top) part of the striatum (Avena & Bocarsly, 2012).

Although we can expect further neurotransmitter research in the future, keep in mind that much of this work focuses on brain mechanisms relevant to hunger, eating and satiety (and much of it has been carried out in animals) but does little to account for other key features of eating disorders, in particular the intense fear of gaining weight. Furthermore, as suggested, the evidence so far does not show that brain changes predate the onset of eating disorders. Thus, we know that brain activity or gene expression of certain dopamine genes is correlated with eating disorders, not that these factors cause eating disorders.

## Cognitive–behavioural factors

Cognitive–behavioural theories of eating disorders focus on understanding the thoughts, feelings and behaviours that contribute to distorted body image, fear of weight gain and loss of control over eating. People with eating disorders may have maladaptive schemata that narrow their attention towards thoughts and images related to weight, body shape and food (Fairburn, Shafran, & Cooper, 1999).

### Anorexia nervosa

Cognitive–behavioural theories of anorexia nervosa emphasise fear of weight gain and body-image disturbance as the motivating factors that powerfully reinforce weight loss. People who develop anorexia nervosa symptoms report that the onset followed a period of weight loss and dieting. Behaviours that achieve or maintain thinness are negatively reinforced by the reduction of anxiety about gaining weight as well as positively reinforced by comments from others (‘Did you lose weight? You look great!’). Dieting and weight

loss may also be positively reinforced by the sense of mastery or self-control they create (Fairburn et al., 1999). Some theories also include personality and sociocultural variables in an attempt to explain how fear of fatness and body-image disturbances develop and will be maintained (Treasure & Schmidt, 2013). For example, perfectionism and a sense of personal inadequacy may lead a person to become especially concerned with his or her appearance, making dieting a potent reinforcer. Similarly, seeing portrayals in the media of thinness as an ideal and tending to compare oneself with especially attractive others all contribute to dissatisfaction with one's body (Hargreaves & Tiggemann, 2003; Tiggemann & Slater, 2004).

Another important factor in producing a strong drive for thinness and a disturbed body image is criticism from peers and parents about being overweight. In one study supporting this conclusion, adolescent boys and girls aged 11 to 15 were evaluated twice, with a one-year interval between assessments. Obesity at the first assessment was related to pressure to be thin perceived from friends or family; at the second assessment, it was linked to dissatisfaction with weight and shape (Stice & Whitenton, 2002). Another study, asking young adults in Singapore about comments their parents had made about their weight and shape when they were younger, revealed that negative comments by their mothers were associated with higher rates of body dissatisfaction and disordered eating for both males and females (Chng & Fassnacht, 2016).

Binge eating often occurs when diets are broken (Polivy & Herman, 1985). Thus, when a person with anorexia nervosa experiences a lapse in a strict diet, the lapse is likely to escalate into binge eating. The purging after an episode of binge eating can again be seen as motivated by the fear of weight gain elicited by the binge-eating episode.

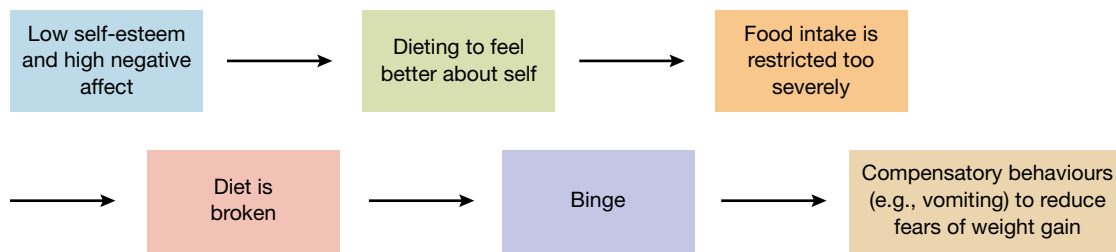
Research has also examined the role of emotion in anorexia nervosa. Not surprisingly, people with anorexia nervosa experience many negative emotions. However, people with anorexia nervosa also experience positive emotion, even though they may not distinguish among different positive emotional states all that well (Selby et al., 2015). In other words, they may experience a positive emotion such as pride very intensely after losing weight or by avoiding eating a piece of cake at a party. This may in turn be indistinguishable from happiness or success and is referred to as low positive emotion differentiation. One study found that low positive emotion differentiation prospectively predicted eating disorder behaviours such as vomiting, checking weight, exercising excessively and using laxatives in a group of 118 women with anorexia nervosa (Selby et al., 2014). Feeling more strong negative emotions also predicted these behaviours.

### **Bulimia nervosa and binge-eating disorder**

People with bulimia nervosa are also thought to be overconcerned with weight gain and body appearance; indeed, they often view their self-worth in terms of their weight and shape. They also have low self-esteem, and because weight and shape are somewhat more controllable than other features of the self, they tend to focus on weight and shape, hoping their efforts in this area will make them feel better generally. They try to follow a very rigid pattern of restrictive eating, with strict rules regarding how much to eat, what kinds of food to eat and when to eat. These strict rules are inevitably broken and the lapse escalates into a binge. After the binge, feelings of disgust and fear of becoming fat build up, leading to compensatory actions such as vomiting (Byrne & McLean, 2002b; Fairburn, 1997). Although purging temporarily reduces the anxiety from having eaten too much, this cycle lowers the person's self-esteem, which triggers more binge eating and purging to maintain desired body weight, which then leads to serious medical consequences (see figure 8.3 for a summary of this theory).

One group of researchers developed the Restraint Scale (see table 8.6), a questionnaire measuring concerns about dieting and overeating (Herman & Polivy, 1980). The researchers have conducted a series of laboratory studies, typically under the guise of being taste tests, with people who scored high on this measure. One such study was described as an assessment of the effects of temperature on taste (Polivy, Heatherton, & Herman, 1988). To achieve a 'cold' condition, some participants first drank a 15-ounce (approximately 440 mL) chocolate milk shake (termed a *preload* by the investigators) and were then given three bowls of ice-cream to taste and rate for flavour. Participants were told that once they had completed their ratings, they could eat as much of the ice-cream as they wanted. The researchers then measured the amount of ice-cream eaten.

**FIGURE 8.3** Schematic of cognitive-behavioural theory of bulimia nervosa



In laboratory studies following this general design, people who scored high on the Restraint Scale ate more than non-dieters after a fattening preload, even when the preload was perceived as fattening but was actually low in calories (Polivy, 1976) and even when the food was relatively unpalatable (Polivy, Herman, & McFarlane, 1994). Thus, people who score high on the Restraint Scale show a pattern similar to that of people with bulimia nervosa, albeit at a much less intense level.

Several additional conditions have been found to further increase the eating of restrained eaters after a preload, most notably various negative mood states, such as anxiety and depression. The increased consumption of people who restrain their eating is especially pronounced when their self-image is threatened (Herman, Polivy, Lank, & Heatherton, 1987) and if they have low self-esteem (Polivy et al., 1988). Further, when people who restrain their eating are given false feedback indicating that their weight is high, they respond with increases in negative emotion and increased food consumption (McFarlane, Polivy, & Herman, 1998). In addition, experimentally induced negative mood has been shown to result in overeating (Chua et al., 2004).

The eating pattern of people with bulimia nervosa or binge-eating disorder is similar to, but more extreme than, the behaviour highlighted in the studies of people who restrain their eating. People with bulimia nervosa or binge-eating disorder typically binge when they encounter stress and experience negative emotions. Using ecological momentary assessments (EMA; see the chapter on diagnosis and assessment), investigators are able to show how specific binge-eating and purging events are linked to changes in emotions and stress in the course of daily life. A meta-analysis of 36 EMA studies found that negative affect preceded the onset of a binge among people with bulimia nervosa or binge-eating disorder, but the effect sizes were stronger for binge-eating disorder than for bulimia nervosa (Haedt-Matt & Keel, 2011). The binge may therefore function as a means of regulating negative affect. However, the meta-analysis also showed that people with bulimia nervosa or binge-eating disorder experienced *more* negative affect after the binge, so the use of binge eating as a way to regulate affect appears not to be successful. Evidence also supports the idea that stress and negative affect are relieved by purging. That is, negative affect levels decline and positive affect levels increase after a purge event, supporting the idea that purging is reinforced by negative affect reduction (Haedt-Matt & Keel, 2011).

Given the similarities between people who score high on the Restraint Scale and people with bulimia nervosa, restrained eating might play a central role in bulimia nervosa. In fact, a study of the naturalistic course of bulimia nervosa (i.e., the course of bulimia nervosa left untreated) has found that the relationship between concern over shape and weight and binge eating was partially mediated by restrained eating (Fairburn et al., 2003). In other words, concerns about body shape and weight predicted restrained eating, which in turn predicted an increase in binge eating across five years of follow-up assessments. However, other studies have failed to find this relationship (Byrne & McLean, 2002b), thus additional research will need to sort out the ways in which restraint eating is linked with symptoms of bulimia nervosa.

Research methods from cognitive science have been used to study how attention, memory and problem solving are impacted in people with eating disorders. Using cognitive tasks such as the Stroop task (see chapter 1) and the dot probe test (see the chapter on anxiety), research shows that people with anorexia nervosa and bulimia nervosa focus their attention on food-related words or images more

than other images (Brooks, Prince, Stahl, Campbell, & Treasure, 2011). People with anorexia nervosa and people who score high on restrained eating remember food words better when they are full but not when they are hungry (Brooks et al., 2011). Other studies investigating cognitive biases to appearance-related stimuli in undergraduate student populations revealed that individuals with stronger body dissatisfaction allocate more attention to their own body areas they dislike and the body parts of others they like (Rodgers & DuBois, 2016). Interestingly, the induction of attentional biases towards shape/weight-related information can also lead to increased body dissatisfaction (Smith & Rieger, 2006). Thus, both women with body dissatisfaction and/or eating disorder symptoms pay greater attention not only to their own bodies, food and weight but also to other women's bodies, food and shapes, which in turn leads to greater body dissatisfaction. This bias towards food and body image may make it harder for women with eating disorders to change their thinking patterns. As we shall see later, cognitive-behavioural therapy devotes time to teaching people with eating disorders to alter these memory and attention biases. Sociocultural factors appear to play a role in the faulty perceptions and eating habits of those with eating disorders. We turn to these influences next.

## Sociocultural factors

Throughout history, the standards societies have set for the ideal body — especially the ideal female body — have varied greatly. Think of the famous nudes painted by Rubens in the seventeenth century: according to modern standards, these women are chubby. Over the past decades, the Western cultural ideal has progressed steadily towards increasing thinness. For example, *Playboy* centrefolds over the years became thinner and thinner; a study describing the anthropomorphic characteristics of 559 *Playboy* models from 1953 to 2003 found a gradual decrease in BMI: centrefold models on average increased in height while weight remained constant (Seifert, 2005).

For men, the situation appears somewhat different. In a study parallel to the study examining *Playboy* centrefolds, researchers analysed the BMI of *Playgirl* male centrefolds from 1973 to 1997 (Leit, Pope, & Gray, 2001). They found that the centrefolds' BMI *increased* over the period and that their muscularity, assessed using a fat-to-muscle estimate, increased even more. Thus, for men the desirable ideal body combines low body fat with muscularity (especially, broad shoulders and big biceps); both of these aspects need to be considered and assessed in the conceptualisation of male body dissatisfaction (Griffiths, Murray, & Touyz, 2013).

Somewhat paradoxically, as cultural standards were moving in the direction of thinness over the latter part of the twentieth century, more and more people were becoming overweight. The prevalence of obesity has more than doubled since 1980 (see focus on discovery 8.1). Currently, almost two-thirds of Australians are overweight or obese, setting the stage for greater conflict between the cultural ideal and reality.

As society has become more health and fat conscious, dieting to lose weight is common; two large Australian studies reported that almost half of their participants were trying to lose weight or were on a diet (Kenardy, Brown, & Vogt, 2001; Timperio, Cameron-Smith, Burns, & Crawford, 2000). Results from a survey of over 4000 women in the United States between the ages of 25 and 45 indicated that a third of the respondents reported spending over half of their lifetimes trying to lose weight (Reba-Harrelson et al., 2009). However, long-term weight loss is hard to achieve and maintain. For example, a recent systematic review evaluating commercial weight-loss programs in the United States reported only small to moderate long-term weight loss, lacking evidence and sometimes even potential risks for some interventions (Gudzune et al., 2015). Every year there are new weight-loss trends and the internet is full of fad diets. But are some diets more effective than others? A study in the *New England Journal of Medicine* reported that diets were equally effective in the short term (i.e., six months), whether carbs, fat or protein were cut, as long as the total number of calories is reduced (Sacks et al., 2009). Surgeries such as liposuction (vacuuming out fat deposits just under the skin) and gastroplasty (surgically changing the stomach so that it cannot digest as much food) remain popular despite their risk (Brownell & Horgen, 2004). However, evidence suggests that interventions for severely obese individuals, such as bariatric surgery, are effective in improving health outcomes (Kopelman & Caterson, 2009).

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Former Treasurer of Australia Joe Hockey had a gastric sleeve fitted in order to accomplish weight loss.



The numbers above indicate that women are more likely than men to be on a diet. The onset of eating disorders is typically preceded by dieting. A large longitudinal study in Victoria, Australia, found that dieting is the most important predictor of eating disorders onset, especially for adolescent girls (Patton, Selzer, Coffey, Carlin, & Wolfe, 1999). Often concerns about weight and shape lead people to diet, supporting the idea that social standards stressing the importance of thinness play a role in the development of these disorders (Striegel-Moore & Bulik, 2007).

It is likely that women who are either overweight or afraid of being fat are also dissatisfied with their bodies. Not surprisingly, studies have found that adolescents with both a high BMI and body dissatisfaction are at higher risk for developing eating disorders (Neumark-Sztainer, Paxton, Hannan, Haines, & Story, 2006; Ricciardelli & McCabe, 2001). Preoccupation with being thin or feeling pressure to be thin predicts an increase in body dissatisfaction among adolescent girls, which in turn predicts more dieting, eating pathology and negative emotions (Paxton et al., 1991; Thompson & Stice, 2001); these factors were also operating in the clinical case of Jing, presented earlier.

Finally, exposure to media portrayals of unrealistically thin models can influence reports of body dissatisfaction. A large meta-analysis of 77 studies investigating the link between media exposure and women's body image concerns showed, perhaps not surprisingly, that exposure to media images depicting the thin-ideal body was related to greater body image concerns for women (Grabe, Ward, & Hyde, 2008). Another study found that men's body dissatisfaction was greater after watching brief video clips of a muscular singer; however, the study only reported this negative effect for younger men (aged 18–30) compared to middle-aged or older men (Mulgrew & Cragg, 2015).

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Cultural standards regarding the ideal feminine shape have changed over time. Even in the 1950s and 1960s, the feminine ideal was considerably heavier than what it has become in the 1970s through to today.



The sociocultural ideal of thinness is a likely vehicle through which people often learn to fear being or even feeling fat and this was probably influential in the cases of both Emily and Jing. In addition to creating an undesired physical shape, being fat is stigmatised and has negative connotations, such as being unsuccessful and having little self-control. Obese people are viewed by others as less smart and are stereotyped as lonely, shy and greedy for the affection of others (Puhl & Heuer, 2009). Even more disturbing, health professionals who specialise in obesity have also exhibited beliefs that obese people are lazy, stupid or worthless (Tomiya et al., 2015). A recent study exploring obesity stigma in Singapore revealed that such attitudes are also present among Asian females; however, they are often not expressed explicitly (Jiang, Tan, & Fassnacht, 2016). Reducing the stigma associated with being overweight will be beneficial to those with eating disorders as well as those who are obese.

Not only does the fear of being ‘fat’ contribute to eating pathology, but the celebration of extreme thinness via websites, blogs and magazines may also play a role. Websites that are ‘pro-ana’ (short for anorexia) or ‘pro-mia’ (short for bulimia) and other ‘thinspiration’ websites, blogs and Instagram profiles are dangerous online communities that promote and exacerbate eating-disorder symptoms and behaviours. These sites often post photos of female celebrities who are extremely thin as inspiration (hence, the term *thinspiration*). Some of these women have publicly discussed their struggles with eating disorders (e.g., the actor Portia de Rossi), but others have not.

A recent review of the impact of these ‘pro-eating disorder’ websites noted that women who visited these sites were more dissatisfied with their bodies and had more eating disorder symptoms (Rouleau & von Ranson, 2011). To tease apart causation from correlation, researchers have randomly assigned healthy women to view either pro-eating disorder, other health-related or tourist websites (Jett, LaPorte, & Wanchis, 2010), supposedly as part of a website evaluation survey. Women completed food diaries for one week before and one week after viewing these websites. Women assigned to the pro-eating disorder website condition restricted their eating more the following week than did the women assigned to the other website conditions. These results suggest that viewing these websites has the potential to cause unhealthy changes in eating behaviour.

#### RESEARCH EXAMPLE

##### Eating disorders and Facebook use

Tiggemann and Slater (2013) assessed 1087 Australian adolescent girls, aged 13–15 years, to investigate the relationship between internet exposure (e.g., use of Facebook) and body image concerns. This study adds to the limited understanding of how internet exposure and, in particular, the use of popular social-networking sites, might lead to body image concerns in adolescents. The authors of this study found 75 percent of the girls had a Facebook profile with an average of 214 ‘friends’, and almost all girls spent on average about two hours online. Time spent online was significantly associated with body image concerns, such as the drive for thinness. In addition, Facebook users had greater body image concerns compared to non-users, and having more Facebook friends was significantly related to greater body image concerns. One of the major limitations of this study, however, is the correlational design, which does not allow for any causal conclusions as to whether using Facebook will lead to greater body image concerns. This study has an important implication for media literacy programs that could educate adolescent girls to become more critically aware of the idealised images presented online and on social-networking sites.

##### QUESTION

Can you conclude from the Tiggemann and Slater (2013) study that using Facebook will lead to greater body image concerns in adolescent girls?

#### Gender influences

We have discussed the fact that eating disorders are more common in women than in men, even though that does not mean that eating disorders in males do not exist. One primary reason is likely the fact that Western cultural standards emphasise and reinforce the desirability of being thin more for women than for men. The risk for eating disorders appears to be especially high in models, dancers and gymnasts, as

in the case of Jing — a group of women who might be particularly concerned with thinness and weight (Byrne & McLean, 2002a).

Another sociocultural factor has remained remarkably resilient to change — namely, the objectification of women's bodies. Women's bodies are often viewed through a sexual lens; in effect, women are defined by their bodies, whereas men are esteemed more for their accomplishments, although men are also reporting increasingly objectification of their bodies (Slater & Tiggemann, 2010). According to the objectification theory (Fredrickson & Roberts, 1997), the prevalence of objectification messages in Western culture (e.g., in television, advertisements and so forth) has led some women to 'self-objectify', which means that they see their own bodies through the eyes of others. Research has shown that self-objectification causes women to feel more shame about their bodies. Shame is most often elicited in situations where an individual's ideal falls short of a cultural ideal or standard. Thus, women are likely to experience body shame when they observe a mismatch between their ideal self and the cultural (objectified) view of women. Research has also shown that both self-objectification and body shame are associated with disordered eating (for a recent review, see Tiggemann, 2013).

Do eating disorders and weight concerns go away as women get older? A large, 20-year prospective study of over 600 men and women reported important differences in dieting and other eating disorder risk factors for men and women over time (Keel, Baxter, Heatherton, & Joiner, 2007). The men and women were first surveyed about dieting, weight, body image and eating disorder symptoms when they were in college. Follow-up surveys were completed 10 and 20 years after college. Thus, the men and women were around age 40 at the 20-year follow-up assessment. The researchers found that after 20 years, women dieted less and were less concerned about their weight and body image than they were in college, even though they actually weighed more now. In addition, eating disorder symptoms decreased over the 20 years for women, as did the risk factors for eating disorders (concern about body image, frequency of dieting). Changes in life roles — having a life partner, having a child — were also associated with decreases in eating disorder symptoms for women. Like women, men weighed more in their early 40s compared to their college time, but in contrast they were now more concerned about their weight and were dieting more. Decreases in risk factors such as concern about body image and dieting frequency were also associated with decreases in eating disorder symptoms for men. However, this might be only one side of the story: one of the few studies investigating eating disorder prevalence rates in women in midlife found that both anorexia nervosa and bulimia nervosa were common, both due to new onset and chronic cases (Micali et al., 2017). Further research is needed to examine eating disorder rates in midlife and older women.

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Celebrities such as Portia de Rossi have publicly discussed their struggles with eating disorders.



### **Cultural factors and ethnic background**

Cultural factors are crucial to consider when describing, diagnosing and treating mental health conditions in general and eating disorders specifically. Historically described as conditions that predominantly affect

adolescent Caucasian females in Western societies, today eating disorders are global mental health conditions. As described above, the importance of a female beauty ideal in Western cultures has historically influenced our understanding of the aetiology and maintenance of eating disorders. However, we have recently seen increasing prevalence rates of eating disorders in non-Western countries such as Singapore (Ho, Tai, Lee, Cheng, & Liow, 2006), Japan (Hotta et al., 2015) and China (Tong et al., 2014), which are often on a par with rates in Western regions such as the United States, the United Kingdom and Australia. An important caveat must be made, however: the form of anorexia observed in non-Western cultures does not always include the intense fear of gaining weight or being fat that is part of the DSM criteria. Thus, intense fear of fat likely reflects an ideal more widely espoused in Westernised cultures. For example, Lee (1991) described a disorder similar to anorexia nervosa in Hong Kong that involves severe emaciation, food refusal and amenorrhoea, but not a fear of becoming fat. Is this a cultural variant of anorexia nervosa or a different disorder? This question is one of the challenges that is faced by cross-cultural researchers (Lee, Lee, Ngai, Lee, & Wing, 2001). Indeed, in some other cultures, higher weight among women is especially valued and considered a sign of fertility and healthiness (Swami et al., 2010).

The variation in the clinical presentation of anorexia nervosa across cultures provides a window into the importance of culture in establishing realistic versus potentially disordered views of one's body. However, there is also evidence that cultural variation is diminishing when it comes to eating disorders. A 20-year study of eating disorders in Hong Kong found evidence of Western influence in both the prevalence and presentation of eating disorders (Lee, Ng, Kwok, & Fung, 2010). First, both anorexia nervosa and bulimia nervosa were twice as common in 2007 than they were in 1987. Second, 25 percent more women reported body dissatisfaction and fear of fat in 2007 than in 1987. Thus, in a fairly short period of time, both body image concerns and eating disorders in Hong Kong appear to have become matched to their Western counterparts.

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Standards of beauty vary cross-culturally, as shown by Gauguin's painting of Tahitian women.



Why are eating disorders in non-Western societies on the rise? A popular explanation is that these countries are becoming more ‘Westernised’ by adapting Western ideas and cultural norms. However, a recent review of the literature exploring this question for Asian countries suggests that this analysis may be incomplete (Pike & Dunne, 2015). Instead, the authors propose that changes in Asian societies due to an increasing industrialisation and urbanisation ‘mirror’ the rise of eating disorder in Asian countries; more developed economies such as Japan, Hong Kong and Singapore report similar numbers of eating disorders compared to their Western counterparts, whereas less developed, poorer countries such as Cambodia, Myanmar or Fiji seem to have less people suffering from eating disorders. But this might be changing with increasing industrialisation and urbanisation of these countries.

The importance of considering cultural factors in the development of eating disorders is demonstrated in a series of studies conducted in the late nineties in Fiji, a society where traditionally fuller bodies are valued as a sign of health. When in 1995 Western television programs were introduced into the previously media-naïve Fijian population, Anne Becker and her team from Harvard Medical School conducted interviews assessing eating disorder behaviours, such as binge eating and purging, with adolescent Fijian girls. Three years later, after prolonged exposure to Western television, eating disorder behaviours had gone up significantly; for example, self-induced vomiting to control weight had increased from 0 percent to over 11 percent (Becker, Burwell, Gilman, Herzog, & Hamburg, 2002). Many girls expressed body concerns as a means of modelling themselves after television characters, leading them to become interested in losing weight. Furthermore, acculturation — the extent to which someone assimilates their own culture with a new culture — may be another important variable to consider. This process can at times be quite stressful. For example, an Australian study found that less-acculturated Asian girls appeared to have unhealthier attitudes and psychopathology towards eating compared to Caucasian and more acculturated Asian girls (Jennings, Forbes, McDermott, Juniper, & Hulse, 2005).

Little is known about eating disorders in Indigenous populations. However, one Australian epidemiological study investigating eating disorders in Australian Aboriginal and Torres Strait Islander peoples found that eating disorder symptoms are at least as frequent as in non-Indigenous Australians (Hay & Carriage, 2012). Although the authors advise interpreting the findings with caution due to a small sample, Indigenous Australians seem to have similar weight and shape concerns and eating disordered behaviour as non-Indigenous Australians. This is confirmed by another study, which compared levels of body dissatisfaction among Indigenous and non-Indigenous Australian adolescents; not surprisingly, girls were generally more dissatisfied with their bodies than boys, but Indigenous adolescents in general reported more strategies to lose weight than did non-Indigenous adolescents (McCabe, Ricciardelli, Mellor, & Ball, 2005).

## Other factors contributing to the aetiology of eating disorders

### Personality influences

We have already seen that neurobiological changes occur as a result of eating disorders. It is also important to keep in mind that an eating disorder itself can affect personality. A study of semistarvation in male conscientious objectors conducted in the late 1940s supports the idea that the personality of people with eating disorders, particularly those with anorexia nervosa, is affected by their weight loss (Keys, Brozek, Henschel, Mickelsen, & Taylor, 1950). The Minnesota Starvation Experiment included 36 healthy young men, who were given two meals a day, totalling 1500 calories over a period of six weeks, to simulate the meals in a concentration camp during World War II. On average, the men lost 25 percent of their body weight. They all soon became preoccupied with food; they also reported increased fatigue, poor concentration, lack of sexual interest, irritability, moodiness and insomnia. Four became depressed and one developed bipolar disorder. This research shows vividly how severe restriction of food intake

can have powerful effects on personality and behaviour, which we need to consider when evaluating the personalities of people with anorexia nervosa and bulimia nervosa.

In part as a response to the findings just mentioned, researchers have studied the relationship between personality and eating disorders. Being overly perfectionistic has been found to be a risk and maintenance factor for both anorexia nervosa and bulimia nervosa (Stice, 2002); however, it seems not to be specific to eating disorders but also relevant to, for example, anxiety disorders and depression (Egan, Wade, & Shafran, 2011). There is also a strong overlap between obsessive–compulsive disorder and anorexia nervosa, which is at least in part due to shared genetic factors (Cederlöf et al., 2015). The description of people with bulimia nervosa often includes the additional characteristics of histrionic features such as impulsivity and sensation seeking (Cassin & von Ranson, 2005).

### Characteristics of families

It is important to understand that family characteristics can play a role in the aetiology and maintenance of eating disorders; however, it is overly simplistic and wrong to assume that family factors are seen as primary causes for these disorders and to blame parents for their children's conditions (Le Grange, Lock, Loeb, & Nicholls, 2010).

Studies of the characteristics of families of people with eating disorders are scarce and have yielded variable results. Some of the variation stems, in part, from the different methods used to collect the data (e.g., retrospectively) and from the sources of the information. For example, retrospective self-reports of people with eating disorders frequently reveal high levels of conflict in the family (e.g., Bulik et al., 2000). Reports of parents, however, do not necessarily indicate high levels of family problems. One way of assessing effects of family characteristics or parenting in the prediction of later onsets of eating disorders are prospective, longitudinal studies. As these studies are hard to conduct, few exist, and their results are not consistent: while one study found that maladaptive parental behaviour (e.g., guilt to control the child or verbal abuse) during childhood increased the risk for eating disorders during adolescence or early adulthood (Johnson, Cohen, Kasen, & Brook, 2002), another study failed to identify the role of parenting as a factor increasing the risk for an eating disorder at the age of 30 (Nicholls & Viner, 2009).

Another way of shedding light on the role of family functioning is to directly study family interactions using observational measures rather than self-reports alone. Although an adolescent's perception of his or her family's characteristics is important, we also need to know how much of reported family discord is perceived and how much is consistent with others' perceptions. In one of the few observational studies conducted thus far, adolescent girls with anorexia nervosa were observed while they interacted with their mothers during a high-conflict family problem-solving task. Daughters with anorexia nervosa and their mothers showed both more disagreement, blame, mind-reading and negative affect compared to other female psychiatric patients and their mothers (Lattimore, Wagner, & Gowers, 2000). While observational studies such as this, coupled with data on perceived family characteristics, are important to help determine whether actual or perceived family characteristics are related to eating disorders, they are challenging to conduct.

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Severe food restriction can have profound effects on behaviour and personality, as illustrated by the study of male conscientious objectors in the 1940s.



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Some people with eating disorders report that their family life was high in conflict.



While parents should not be blamed or made responsible for their children's eating disorder, it is important to educate them that negative comments about the child's weight and physical appearance can lead to body dissatisfaction and disordered eating behaviours (Rodgers, Paxton, & Chabrol, 2009). For example, in a large population-based sample of boys and girls from Western Australia, parents' perceptions of their child being overweight at age 8 or 10 years was a strong factor in predicting eating disorders in children aged 14 years (Allen, Byrne, Forbes, & Oddy, 2009).

### **Child abuse and eating disorders**

Studies have consistently found that child sexual abuse leads to an increased risk for mental disorders (Spataro, Mullen, Burgess, Wells, & Moss, 2004). An epidemiological study from New Zealand explored effects of child sexual abuse on later eating disorders and found that women who had experienced sexual abuse as a child developed anorexia nervosa or bulimia nervosa more often than women who reported no abuse. However, women who have been sexually abused as children are also more likely to develop depression and anxiety, which suggests that sexual abuse during childhood makes individuals more vulnerable for mental disorder in general, not only eating disorders. In addition, other forms of child abuse such as physical and emotional abuse and neglect, also contribute to the later development of mental health problems and eating disorders (Norman et al., 2012). However, more studies are needed to examine why some people who have experienced maltreatment develop anxiety disorders or depression whereas others experience eating disorders, and also whether specific forms of

maltreatment are associated with certain types of body concerns or eating disorders (e.g., body image concerns or binge eating).

## 8.3 Treatment of eating disorders

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**LEARNING OUTCOME 8.3** Describe the treatments for eating disorders and the evidence supporting their effectiveness.

Hospitalisation is frequently required to treat people with anorexia nervosa so that their ingestion of food can be gradually increased and carefully monitored. For example, this was necessary for Emily. Weight loss can be so severe that tube feeding (e.g., nasogastric feeding through the nose) is necessary to save the person's life. The medical complications of anorexia nervosa, such as electrolyte imbalances, also require treatment. For anorexia nervosa, bulimia nervosa and binge-eating disorders, both psychological treatments and medications have been used.

### Psychological treatment of anorexia nervosa

There are a number of different psychological interventions to choose from when treating individuals suffering from anorexia nervosa. However, major shortcomings in the existing studies make it impossible to directly compare commonly used psychological treatments, leaving it unclear what the best treatment option is (Hay, Claudino, Touyz, & Abd Elbaky, 2015). In the following, the most promising psychotherapeutic approaches to this life-threatening disorder will be presented.

Therapy for anorexia nervosa is generally believed to be a two-step process. The immediate goal is to help the person gain weight in order to avoid medical complications and the possibility of death. The person is often so weak and the physiological functioning so disturbed that hospital treatment is necessary. When patients have been malnourished over long periods, reinstitutions of nutrition can lead to the so-called refeeding syndrome, a dangerous and sometimes fatal metabolic disturbance (e.g., due to very low levels of potassium, phosphate and magnesium). Thus, refeeding guidelines suggest starting with low doses of nutrition, increasing slowly over time while constantly monitoring the patient's electrolyte levels. Beyond the immediate medical stabilisation of a patient, the major goal of treatment — long-term maintenance of weight gain — remains a challenge.

Psychological treatments for anorexia nervosa can be divided into individual and family-based interventions. Among the individual therapies, cognitive-behavioural therapy (CBT), especially the CBT-Enhanced format developed at the University of Oxford (Fairburn et al., 2008), shows promising results (Fairburn et al., 2013). CBT focuses primarily on the maintaining processes of anorexia nervosa by directly challenging cognitions and patterns of thinking (e.g., cognitive biases such as 'People only like thin women!'). Different treatment studies suggest that CBT is an effective treatment for anorexia nervosa (Hay, Claudino, Touyz, & Elbaky, 2015); around 60–70 percent of patients show significant weight gains and reductions in eating disorder psychopathology. However, one-third of patients do not respond to treatment and some of these individuals develop chronic, severe and enduring anorexia nervosa. Specialist supportive clinical management (SSCM), which primarily focuses on the resumption of normal eating and restoration of weight, is another recommended treatment option for adults with anorexia nervosa. A recent randomised controlled clinical trial compared a modified version of CBT with SSCM in a sample of women with enduring and severe anorexia nervosa and found that they were equally effective in reducing eating disorder symptoms and depression (Touyz et al., 2013). Although BMI increased at the end of treatment and remained stable at the 12-month follow-up, the average BMIs for women in both treatment groups were still in the mild severity range (see table 8.2).

A randomised controlled clinical trial conducted in Germany compared CBT to psychodynamic therapy and to 'optimised treatment as usual', which included support from general medical practitioners and referrals to psychotherapists (Zipfel et al., 2014). All three treatments were equally effective in increasing BMI, but again the increase was only modest: for example, the CBT group had gained on average about 1.6 BMI points at the 12-month follow-up.

Family therapy is another form of psychological treatment for anorexia nervosa and is based on the notion that interactions among members of the patient's family can play a role in treating the disorder (Lock & Le Grange, 2005). A family-based therapy (FBT) developed at the Maudsley Hospital in London focuses on helping parents restore their child to a healthy weight while at the same time building up family functioning in the context of adolescent development (Le Grange, 2005). A randomised controlled clinical trial compared FBT with individual therapy and found that both treatments were equally effective at the end of the 24-session treatment. However, more girls receiving FBT were in full remission (49 percent) one year after treatment than girls receiving individual therapy (23 percent) (Lock et al., 2010). Another study of FBT found that the girls who had gained weight by session 4 were more likely to be in full remission at the end of treatment (Doyle, Le Grange, Loeb, Doyle, & Crosby, 2010). Thus, early weight gain may be an important predictor of a good outcome. Although the findings are promising, additional work needs to be done to improve psychological interventions for anorexia nervosa.

## Psychological treatment of bulimia nervosa

CBT is the best-validated and current standard for the treatment of bulimia nervosa (Hay et al., 2014). In CBT, people with bulimia nervosa are encouraged to question society's standards for physical attractiveness. People with bulimia nervosa need to understand that normal body weight can be maintained without severe dieting and that unrealistic restriction of food intake can often trigger binge eating. In CBT, they learn that all is not lost with just one bite of high-calorie food; occasional snacking does not need to trigger binge eating, which is then followed by induced vomiting or taking laxatives resulting in high levels of shame and depression. Altering this all-or-nothing thinking can help people begin to eat more moderately. Patients also learn assertiveness skills, which help them cope with unreasonable demands placed on them by others, as well as more satisfying ways of relating to people.

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Family therapy is a main form of treatment for anorexia nervosa.



The overall goal of treatment in bulimia nervosa is to develop more typical eating patterns. People with bulimia nervosa need to learn to eat three meals a day and even some snacks between meals without sliding back into binge eating and purging. Regular meals control hunger and thereby, it is hoped, the urge to eat enormous amounts of food, which in turn might lead to a reduction in purging. To help people with bulimia nervosa develop less extreme beliefs about themselves, cognitive-behaviour therapists gently but firmly challenge such beliefs as, ‘No one will respect me if I gain weight’. Unrealistic demands and other cognitive distortions — such as the belief that eating a small amount of high-calorie food means that the person is an utter failure and doomed never to improve — are continually challenged. The therapist works collaboratively with the person to identify events, thoughts and feelings that trigger an urge to binge and then to learn more adaptive ways to cope with these situations.

In the case of Jing, she and her therapist discovered that binge eating often took place after she was criticised by her coach. Therapy included the following:

- encouraging Jing to assert herself if the criticism is unwarranted
- desensitising her to social evaluation and encouraging her to question society’s standards for ideal weight and the pressures on women to be thin — not an easy task by any means
- teaching her that it is not a catastrophe to make a mistake and it is not necessary to be perfect, even if the coach’s criticism is valid.

Findings from a number of studies indicate that CBT often results in less frequent binge eating and purging, with reductions ranging from 70 to more than 90 percent. For example, a recent randomised controlled clinical trial compared five months of CBT with two years of psychoanalytic therapy and found that those who received CBT had far fewer binge-eating and purging episodes, both at the end of five months and at the end of two years (Poulsen et al., 2014). Furthermore, CBT reduces significantly extreme dietary restraint and improves attitudes towards body shape and weight (Hay, Bacaltchuk, Stefano, & Kashyap, 2009). Treatment gains are maintained long-term (e.g., in one study up to 10 years; Keel, Mitchell, Davis, & Crow, 2002). However, there are limitations to these positive outcomes. If we focus on the people themselves rather than on the numbers of binge-eating and purging episodes across people, we find that many of those treated with CBT remain symptomatic (Lundgren, Danoff-Burg, & Anderson, 2004). Clearly, while CBT may be the most effective treatment available for bulimia nervosa, there is still room for improvement.

Another form of CBT, called guided self-help CBT, has also shown promise for some people with bulimia nervosa. In this type of treatment, people receive self-help books on topics such as perfectionism, body image, negative thinking, food and health. Patients meet for a small number of sessions with a therapist who helps guide them through the self-help material. Preliminary results suggest that this is an effective treatment compared to a wait-list control group and to traditional CBT for bulimia nervosa. In addition, greater confidence in one’s ability to change is associated with better outcomes (Steele, Bergin, & Wade, 2011). Nonetheless, it is clear that unguided self-help has poorer outcomes compared to guided self-help CBT or face-to-face CBT in bulimia nervosa (Hay, 2013).

Interpersonal therapy (IPT) has also been used for bulimia nervosa and it has fared well in comparisons with CBT, although it has not produced results as quickly (Agras, Walsh, Fairburn, Wilson, & Kraemer, 2000). However, long-term outcomes for CBT and IPT seem to be comparable (Agras et al., 2000). Family therapy is also effective for bulimia nervosa, though it has been studied less frequently than either CBT or IPT. A randomised clinical trial demonstrated that family-based therapy was superior to supportive psychotherapy for adolescents with bulimia nervosa with respect to decreasing binge eating and purging up to six months after treatment was completed (Le Grange, Crosby, Rathouz, & Leventhal, 2007).

## Psychological treatment of binge-eating disorder

Although not as extensively studied as bulimia nervosa, CBT has been shown to be effective for binge-eating disorder in several studies (Vocks et al., 2010). The intervention targets restraint as well as binge

eating by emphasising self-monitoring, self-control and problem solving. Treatment gains from CBT appear to last long-term while IPT seems to be as effective as CBT and seems to have slightly better long-term outcomes than CBT (Hilbert et al., 2012). Both CBT and IPT are more effective than behavioural weight-loss programs, which are often used to treat obesity (Vocks et al., 2010). More specifically, CBT and IPT reduce binge eating (but not necessarily weight), whereas behavioural weight-loss programs may promote weight loss but do not curb binge eating.

## Medications

Medications have also been used to treat anorexia nervosa with little success in improving weight or other core features of anorexia nervosa (de Vos et al., 2014). Because bulimia nervosa is often comorbid with depression, it has been treated with various antidepressants. Findings from most studies, including double-blind randomised controlled trials with placebo controls, confirm the efficacy of a variety of antidepressants (e.g., serotonin reuptake inhibitor, SSRI, such as fluoxetine) in reducing purging and binge eating, even among people who had not responded to prior psychological treatment (Bacaltchuk & Hay, 2003). However, CBT alone is more effective than drug treatment alone; a meta-analysis showed that CBT yielded better results than antidepressant medications (Bacaltchuk, Hay, & Trefiglio, 2001). But are outcomes better when antidepressant medication is added to CBT? Evidence on this front is mixed. Adding antidepressant drugs may be useful in helping to alleviate the depression that often occurs with bulimia nervosa; however, whether the effects of antidepressants on bulimic symptoms are caused by improving depressive symptoms remains unclear (Bacaltchuk, Hay, & Mari, 2000). On the negative side, many people with bulimia nervosa drop out of drug treatment (Bacaltchuk et al., 2001). Moreover, most people relapse when various kinds of antidepressant medications are withdrawn (Bacaltchuk et al., 2000), as is the case with many psychoactive drugs.

Medication treatment for binge-eating disorder has not been well studied. Limited evidence suggests that antidepressant medications are not effective in reducing binge eating or weight loss (Reas & Grilo, 2015). For example, a randomised controlled trial compared the antidepressant medication fluoxetine (Prozac) with CBT plus placebo or CBT plus fluoxetine and found that CBT plus placebo was more effective in reducing binge episodes than either condition including fluoxetine and this remained the case at a 12-month follow-up (Grilo, Crosby, Wilson, & Masheb, 2012).

## Preventive interventions for eating disorders

The best way of treating eating disorders is to prevent them; intervening early before the onset of the disorder may help to prevent these disorders from ever developing. Broadly speaking, three different types of preventive interventions have been developed and implemented.

1. *Psychoeducational approaches.* The focus is on educating children and adolescents about eating disorders in order to prevent them from developing the symptoms.
2. *Deemphasising sociocultural influences.* The focus here is on helping children and adolescents resist or reject sociocultural pressures to be thin.
3. *Risk factor approach.* The focus here is on identifying people with known risk factors for developing eating disorders (e.g., weight and body-image concern, dietary restraint) and intervening to alter these factors.

In a meta-analysis of prevention studies conducted between 1980 and 2006, Stice, Shaw and Marti (2007) found modest support for some prevention approaches. The most effective prevention programs are those that are interactive rather than didactic, target at-risk adolescents aged 15 or older, include girls only and involve multiple sessions rather than just one session. A more recent meta-analysis included 112 articles of randomised controlled trials of eating disorder prevention interventions from 2009 to 2015 (Le, Barendregt, Hay, & Mihalopoulos, 2017). The authors concluded that a number of promising eating disorder prevention programs exist.

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Prevention programs that are interactive have been effective for girls with eating disorders.



Among the best-studied programs is the Body Project, a three-session body-acceptance intervention to help girls and young women to challenge the thin-ideal with activities that are designed to elicit psychological dissonance/discomfort (e.g., verbal, written and behavioural exercises). The program, which was developed at the University of Texas, has been shown to significantly decrease body dissatisfaction and the risk for eating disorder pathology over a three-year follow-up (Stice, Marti, Spoor, Presnell, & Shaw, 2008).

To overcome some of the limitations traditional prevention strategies were facing (e.g., limited reach, high costs), numerous internet-based programs have been developed over the past few years. Such programs offer easily accessible, convenient and anonymous services, which is crucial as many people are ashamed about their problems. A recent meta-analytic review of 20 studies examining the efficacy of internet-based prevention programs for reducing the risk of developing eating disorders reported modest effects for decreasing eating disorder risk factors and symptoms (Melioli et al., 2016). The Student Bodies program, for example, is an eight-week, structured, internet-delivered, psychoeducational intervention based on social learning and cognitive-behavioural theories to improve body image. The program — developed at Stanford University — includes self-monitoring and goal-setting tasks and also online discussion groups (Winzelberg et al., 2000). It has been proven to reduce drive for thinness and negative body image in young females and has been adapted for different populations (e.g., high risk or overweight females) (Beintner, Jacobi, & Taylor, 2012). Another program is Set Your Body Free, which was developed in Australia and consists of eight weekly psychoeducational sessions. A study comparing a face-to-face group version of the program led by a therapist to an online version showed that for adult women the face-to-face delivery was slightly more effective. However, the authors concluded that the online delivery might increase access to help (Paxton, McLean, Gollings, Faulkner, & Wertheim, 2007). Finally, the German-developed prevention and early intervention program ProYouth, which consists of psychoeducation, self-monitoring, peer support and online counselling (Bauer & Moessner, 2013), has been adapted to the Australian context (ProYouth OZ) and is currently being tested.

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## SUMMARY

### **8.1 Describe the symptoms associated with anorexia nervosa, bulimia nervosa and binge-eating disorder and be able to distinguish between the different eating disorders.**

Anorexia nervosa has three characteristics: restriction of energy intake leading to low body weight, an intense fear of gaining weight and being fat, and a distorted body image. Anorexia nervosa usually begins in the early teen years and is more common in women than men. Bodily changes that can occur after severe weight loss can be serious and life threatening. Although approximately 50 percent of women with anorexia nervosa eventually recover, it can take many years.

Bulimia nervosa involves both binge eating and compensatory behaviour. Binge eating often involves sweet foods and is more likely to occur when someone is alone, after encountering stress or experiencing negative emotions. One striking difference between anorexia nervosa and bulimia nervosa is weight loss: people with anorexia nervosa lose a tremendous amount of weight, whereas people with bulimia nervosa alternate between overeating and fasting/purging, but generally do not lose weight. Bulimia nervosa typically begins in late adolescence and is more common in women than men. Depression often co-occurs with bulimia nervosa and each condition appears to be a risk factor for the other. Dangerous changes to the body can also occur as a result of bulimia nervosa, such as menstrual problems, tearing in the stomach and throat, and swelling of the salivary glands. Around 45 percent of people with bulimia nervosa fully recover; CBT is most effective in reducing binge eating and purging as well as changing attitudes towards body shape and weight.

Binge-eating disorder is characterised by frequent binge eating and most (but not all) people who experience the disorder are obese (defined as having a BMI greater than 30). Not all obese people meet the criteria for binge-eating disorder — only those who have binge episodes and report feeling a loss of control over their eating qualify. Binge-eating disorder is more common than anorexia nervosa and bulimia nervosa and occurs more often in women than men, although the gender difference is not as great as it is in anorexia nervosa and bulimia nervosa. About 60 percent of people with binge-eating disorder recover, but it may take even longer than recovery for anorexia nervosa or bulimia nervosa.

### **8.2 Describe the genetic, neurobiological, sociocultural and psychological factors implicated in the aetiology of eating disorders.**

Genetic factors appear to play a role in both anorexia nervosa and bulimia nervosa. Both disorders tend to run in families, and twin studies support the role of genetics in the actual disorders and particular characteristics of the disorders, such as body dissatisfaction, preoccupation with thinness and binge eating. The hypothalamus does not appear to be directly involved in eating disorders and low levels of endogenous opioids are seen concurrently with bulimia nervosa, but not before the onset of the disorder. Thus, changes in food intake could affect the opioid system instead of changes in the opioid system affecting food intake. Serotonin may play a role in eating disorders, with studies finding a decrease in serotonin metabolites in patients with anorexia nervosa and bulimia nervosa. Other research suggests that dopamine may play a role in restrained eating, a characteristic that is found in people with eating disorders. Research linking dopamine to the brain's reward system can help account for how binge eating influences the dopamine system. In summary, neurobiological factors do not explain particularly well some of the key features of anorexia nervosa and bulimia nervosa, in particular the intense fear of becoming fat.

Cognitive-behavioural theories focus on body dissatisfaction, preoccupation with thinness, and attention and memory. Studies have found that concerns about body shape and weight predicted restrained eating, which in turn predicted an increase in binge eating. People with eating disorders pay greater attention to food and body-image-related stimuli and they tend to remember these better as well, suggesting that their attention and memory may be biased towards food and body image.

Sociocultural factors, including society's preoccupation with thinness, may play a role in eating disorders. This preoccupation is linked to dieting efforts, which often precede the development of eating disorders. Preoccupation with thinness and media portrayals of thin women predict an increase in body dissatisfaction, which also precedes the development of eating disorders. The ways in which especially female bodies are objectified may lead some women to self-objectify their bodies, resulting in greater body dissatisfaction and eating pathology.

Although linked to Western female beauty ideals, eating disorders have become global illnesses. Prevalence rates in Asian countries are on the rise, potentially due to industrialisation and urbanisation of these societies. Little research has been done with Indigenous populations, but it seems that body dissatisfaction and eating disorder symptoms are at least as frequent in Australian Aboriginal and Torres Strait Islander peoples as in non-Indigenous Australians.

Lastly, other factors contributing to the development of eating disorders are discussed. Perfectionistic and obsessive-compulsive traits may play a role. Also high rates of sexual and physical abuse are found among people with eating disorders, but these are not risk factors specific to the development of eating disorders.

### **8.3 Describe the treatments for eating disorders and the evidence supporting their effectiveness.**

Therapy for anorexia nervosa must first focus on weight gain. Both CBT and family-based therapies are effective psychological treatments. The most effective psychological treatment for bulimia nervosa is CBT; it involves challenging and changing a patient's beliefs and thinking about thinness, being overweight, dieting and restriction of food, with the overall goal being to re-establish normal eating patterns. Both CBT and IPT seem to be effective treatments for binge-eating disorders; however, more studies are needed.

CBT alone is more effective than medication treatment. Antidepressant medications have shown some benefit in the treatment of bulimia nervosa, but not anorexia nervosa or binge-eating disorder. However, people with bulimia nervosa are likely to discontinue medication or relapse after medication is withdrawn.

Several prevention programs have been effective in decreasing body dissatisfaction and the risk for eating disorder symptoms. More recently, internet-based prevention programs have been developed including psychoeducation, self-monitoring and online counselling.

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## **KEY TERMS**

**anorexia nervosa** a disorder in which a person refuses to maintain normal weight, has an intense fear of gaining weight and feels fat even when emaciated

**binge-eating disorder** included as a disorder in DSM-5; includes recurrent episodes of binge eating

**body mass index (BMI)** measure of body fat calculated by dividing weight in kilograms by height in metres squared; considered a more valid estimate of body fat than many others

**bulimia nervosa** a disorder characterised by recurrent, uncontrollable binge-eating episodes followed by purging either by vomiting or by taking laxatives

**obese** currently defined as exhibiting a body mass index (BMI) of greater than 30

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## **WEBSITES**

1. The Butterfly Foundation is a national non-profit organisation in Australia, which provides a range of eating disorder services such as support groups for people with eating disorder and their carers, telephone and chat support, educational seminars and also financial support for those who cannot afford treatment. (<https://thebutterflyfoundation.org.au>)

2. Eating Disorders Victoria is a state-based service for eating disorders. ([www.eatingdisorders.org.au](http://www.eatingdisorders.org.au))
3. The Centre for Eating and Dieting Disorders is a New South Wales state-based service for eating disorders. (<http://cedd.org.au>)
4. The National Eating Disorders Collaboration (NEDC) is an initiative of the Australian Government Department of Health bringing together people and organisations with expertise or interest in eating disorders. The organisation provides evidence-based information and is open for everyone to join. ([www.nedc.com.au](http://www.nedc.com.au))
5. EDANZ is a support network in New Zealand for people who are suffering from an eating disorder or caring for someone with an eating disorder. Through educational material and advocacy, EDNAZ aims to improve the awareness and understanding of eating disorders in New Zealand. ([www.ed.org.nz](http://www.ed.org.nz))

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## REFERENCES

- Agras, W., Walsh, B., Fairburn, C. G., Wilson, G., & Kraemer, H. C. (2000). A multicenter comparison of cognitive-behavioral therapy and interpersonal psychotherapy for bulimia nervosa. *Archives of General Psychiatry*, 57(5), 459–466. doi: 10.1001/archpsyc.57.5.459
- Albuquerque, D., Stice, E., Rodríguez-López, R., Manco, L., & Nóbrega, C. (2015). Current review of genetics of human obesity: from molecular mechanisms to an evolutionary perspective. *Molecular Genetics and Genomics*, 290(4), 1191–1221. doi: 10.1007/s00438-015-1015-9
- Allen, K. L., Byrne, S. M., Forbes, D., & Oddy, W. H. (2009). Risk factors for full- and partial-syndrome early adolescent eating disorders: a population-based pregnancy cohort study. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(8), 800–809. doi: 10.1097/CHI.0b013e3181a8136d
- Arcelus, J., Mitchell, A. J., Wales, J., & Nielsen, S. (2011). Mortality rates in patients with anorexia nervosa and other eating disorders: A meta-analysis of 36 studies. *Archives of General Psychiatry*, 68(7), 724–731. doi: 10.1001/archgenpsychiatry.2011.74
- Attia, E., Becker, A. E., Bryant-Waugh, R., Hoek, H. W., Kreipe, R. E., Marcus, M. D., . . . Wonderlich, S. (2013). Feeding and eating disorders in DSM-5. *American Journal of Psychiatry*, 170(11), 1237–1239. doi: 10.1176/appi.ajp.2013.13030326
- Attia, E., & Roberto, C. A. (2009). Should amenorrhea be a diagnostic criterion for anorexia nervosa? *International Journal of Eating Disorders*, 42(7), 581–589. doi: 10.1002/eat.20720
- Australian Bureau of Statistics (ABS). (2013). *Australian Health Survey: Updated results, 2011–2012*. Canberra: ABS.
- Australian Bureau of Statistics (ABS). (2015). *National Health Survey: First results, 2014–15*. Canberra: ABS.
- Australian Institute of Health and Welfare (AIHW). (2014). Australia's health 2014. *Australia's health series no. 14*. Canberra: AIHW.
- Avena, N. M., & Bocarsly, M. E. (2012). Dysregulation of brain reward systems in eating disorders: neurochemical information from animal models of binge eating, bulimia nervosa and anorexia nervosa. *Neuropharmacology*, 63(1), 87–96. doi: 10.1016/j.neuropharm.2011.11.010
- Bacaltchuk, J., & Hay, P. (2003). Antidepressants versus placebo for people with bulimia nervosa. *Cochrane Database of Systematic Reviews* (4), CD003391. doi: 10.1002/14651858.cd003391
- Bacaltchuk, J., Hay, P., & Mari, J. J. (2000). Antidepressants versus placebo for the treatment of bulimia nervosa: a systematic review. *Australian and New Zealand Journal of Psychiatry*, 34(2), 310–317. doi: 10.1046/j.1440-1614.2000.00709.x
- Bacaltchuk, J., Hay, P., & Trefiglio, R. (2001). Antidepressants versus psychological treatments and their combination for bulimia nervosa. *Cochrane Database of Systematic Reviews*(4), CD003385. doi: 10.1002/14651858.cd003385
- Bailer, U. F., & Kaye, W. H. (2003). A review of neuropeptide and neuroendocrine dysregulation in anorexia and bulimia nervosa. *Current Drug Targets: CNS and Neurological Disorders*, 2(1), 53–59.
- Bailer, U. F., & Kaye, W. H. (2011). Serotonin: imaging findings in eating disorders. *Current Topics in Behavioral Neurosciences*, 6, 59–79. doi: 10.1007/7854\_2010\_78
- Bauer, S., & Moessner, M. (2013). Harnessing the power of technology for the treatment and prevention of eating disorders. *International Journal of Eating Disorders*, 46(5), 508–515. doi: 10.1002/eat.22109
- Becker, A. E., Burwell, R. A., Gilman, S. E., Herzog, D. B., & Hamburg, P. (2002). Eating behaviours and attitudes following prolonged exposure to television among ethnic Fijian adolescent girls. *British Journal of Psychiatry*, 180, 509–514. doi: 10.1192/bjp.180.6.509
- Beintner, I., Jacobi, C., & Taylor, C. B. (2012). Effects of an internet-based prevention programme for eating disorders in the USA and Germany — A meta-analytic review. *European Eating Disorders Review*, 20(1), 1–8. doi: 10.1002/erv.1130
- Bello, N. T., Patinkin, Z. W., & Moran, T. H. (2011). Opioidergic consequences of dietary-induced binge eating. *Physiology & Behavior*, 104(1), 98–104. doi: 10.1016/j.physbeh.2011.04.032

- Bergen, A. W., Yeager, M., Welch, R. A., Haque, K., Ganjei, J. K., van den Bree, M. B., ... Kaye, W. H. (2005). Association of multiple DRD2 polymorphisms with anorexia nervosa. *Neuropsychopharmacology*, 30(9), 1703–1710. doi: 10.1038/sj.npp.1300719
- Berridge, K. C., & Robinson, T. E. (1998). What is the role of dopamine in reward: hedonic impact, reward learning or incentive salience? *Brain Research: Brain Research Reviews*, 28(3), 309–369. doi: 10.1016/S0165-0173(98)00019-8
- Boffetta, P., McLerran, D., Chen, Y., Inoue, M., Sinha, R., He, J., ... Potter, J. D. (2011). Body mass index and diabetes in Asia: A cross-sectional pooled analysis of 900,000 individuals in the Asia cohort consortium. *PLoS ONE*, 6(6), e19930. doi: 10.1371/journal.pone.0019930
- Boraska, V., Franklin, C. S., Floyd, J. A., Thornton, L. M., Huckins, L. M., Southam, L., et al. (2014). A genome-wide association study of anorexia nervosa. *Molecular Psychiatry*, 19(10), 1085–94.
- Brooks, S., Prince, A., Stahl, D., Campbell, I. C., & Treasure, J. (2011). A systematic review and meta-analysis of cognitive bias to food stimuli in people with disordered eating behaviour. *Clinical Psychology Review*, 31(1), 37–51. doi: 10.1016/j.cpr.2010.09.006
- Brownell, D. K., & Horgen, K. B. (2004). *Food fight: The inside story of the food industry, America's obesity crisis and what we can do about it*. New York: McGraw-Hill.
- Brownley, K. A., Berkman, N. D., Peat, C. M., Lohr, K., Cullen, K. E., Bann, C. M., ... Bulik, M. (2016). Binge-eating disorder in adults: A systematic review and meta-analysis. *Annals of Internal Medicine*, 165(6), 409–420. doi: 10.7326/M15-2455
- Bulik, C. M., & Reichborn-Kjennerud, T. (2003). Medical morbidity in binge eating disorder. *International Journal of Eating Disorders*, 34(S1), S39–S46. doi: 10.1002/eat.10204
- Bulik, C. M., Sullivan, P. F., Wade, T. D., & Kendler, K. S. (2000). Twin studies of eating disorders: A review. *International Journal of Eating Disorders*, 27(1), 1–20. doi: 10.1002/(SICI)1098-108X(200001)27:1<1::AID-EAT1>3.0.CO;2-Q
- Bulik, C. M., Thornton, L., Root, T. L., Pissetsky, E. M., Lichtenstein, P., & Pedersen, N. L. (2010). Understanding the relation between anorexia nervosa and bulimia nervosa in a Swedish national twin sample. *Biological Psychiatry*, 67(1), 71–77. doi: 10.1016/j.biopsych.2009.08.010
- Byrne, S., & McLean, N. (2002a). Elite athletes: Effects of the pressure to be thin. *Journal of Science and Medicine in Sport*, 5(2), 80–94. doi: 10.1016/S1440-2440(02)80029-9
- Byrne, S. M., & McLean, N. J. (2002b). The cognitive-behavioral model of bulimia nervosa: A direct evaluation. *International Journal of Eating Disorders*, 31(1), 17–31. doi: 10.1002/eat.10002
- Cassin, S. E., & von Ranson, K. M. (2005). Personality and eating disorders: A decade in review. *Clinical Psychology Review*, 25(7), 895–916. doi: 10.1016/j.cpr.2005.04.012
- Cederlöf, M., Thornton, L. M., Baker, J., Lichtenstein, P., Larsson, H., Rück, C., ... Mataix-Cols, D. (2015). Etiological overlap between obsessive-compulsive disorder and anorexia nervosa: a longitudinal cohort, multigenerational family and twin study. *World Psychiatry*, 14(3), 333–338. doi: 10.1002/wps.20251
- Chng, S. C. W., & Fassnacht, D. B. (2016). Parental comments: Relationship with gender, body dissatisfaction and disordered eating in Asian young adults. *Body Image*, 16, 93–99. doi: 10.1016/j.bodyim.2015.12.001
- Chua, J. L., Touyz, S., & Hill, A. J. (2004). Negative mood-induced overeating in obese binge eaters: An experimental study. *International Journal of Obesity and Related Metabolic Disorders*, 28(4), 606–610. doi: 10.1038/sj.ijo.0802595
- Cohen, D. A. (2014). *A big fat crisis: the hidden forces behind the obesity epidemic — and how we can end it*. New York: Nation Books.
- Colagiuri, S., Lee, C. M., Colagiuri, R., Magliano, D., Shaw, J. E., Zimmet, P. Z., & Caterson, I. D. (2010). The cost of overweight and obesity in Australia. *Medical Journal of Australia*, 192(5), 260–264.
- Cook, T., Rutishauser, I., & Seelig, M. (2001). *Comparable data on food and nutrient intake and physical measurements from the 1983, 1985 and 1995 national nutrition surveys*. Canberra: CDHAC.
- Crow, S. J., Peterson, C. B., Swanson, S. A., Raymond, N. C., Specker, S., Eckert, E. D., & Mitchell, J. E. (2009). Increased mortality in bulimia nervosa and other eating disorders. *American Journal of Psychiatry*, 166(12), 1342–1346. doi: 10.1176/appi.ajp.2009.09020247
- de Vos, J., Houtzager, L., Katsaragaki, G., van de Berg, E., Cuijpers, P., & Dekker, J. (2014). Meta analysis on the efficacy of pharmacotherapy versus placebo on anorexia nervosa. *Journal of Eating Disorders*, 2, 27. doi: 10.1186/s40337-014-0027-x
- Doyle, P. M., Le Grange, D., Loeb, K., Doyle, A. C., & Crosby, R. D. (2010). Early response to family-based treatment for adolescent anorexia nervosa. *International Journal of Eating Disorders*, 43(7), 659–662. doi: 10.1002/eat.20764
- Eddy, K. T., Keel, P. K., Dorer, D. J., Delinsky, S. S., Franko, D. L., & Herzog, D. B. (2002). Longitudinal comparison of anorexia nervosa subtypes. *International Journal of Eating Disorders*, 31(2), 191–201. doi: 10.1002/eat.10016
- Egan, S. J., Wade, T. D., & Shafran, R. (2011). Perfectionism as a transdiagnostic process: A clinical review. *Clinical Psychology Review*, 31(2), 203–212. doi: 10.1016/j.cpr.2010.04.009
- Fairburn, C. G. (1997). Eating disorders. In D. M. Clark & C. G. Fairburn (Eds.), *Science and practice of cognitive behavior therapy* (pp. 209–243). New York: Oxford University Press

- Fairburn, C. G., Cooper, Z., Doll, H. A., O'Connor, M. E., Palmer, R. L., & Dalle Grave, R. (2013). Enhanced cognitive behaviour therapy for adults with anorexia nervosa: A UK–Italy study. *Behaviour Research and Therapy*, 51(1), R2–R8. doi: 10.1016/j.brat.2012.09.010
- Fairburn, C. G., Cooper, Z., Shafran, R., Bohn, K., Hawker, D., & Murphy, R. (2008). Enhanced cognitive behavior therapy for eating disorders: the core protocol. In F. C. G. (Ed.), *Cognitive behavior therapy and eating disorders* (pp. 47–193). New York: Guilford Press
- Fairburn, C. G., Doll, H. A., Welch, S. L., Hay, P. J., Davies, B. A., & O'Connor, M. E. (1998). Risk factors for binge eating disorder: a community-based, case-control study. *Archives of General Psychiatry*, 55(5), 425–432. doi: 10.1001/archpsyc.55.5.425
- Fairburn, C. G., Shafran, R., & Cooper, Z. (1999). A cognitive behavioural theory of anorexia nervosa. *Behaviour Research and Therapy*, 37(1), 1–13.
- Fairburn, C. G., Stice, E., Cooper, Z., Doll, H. A., Norman, P. A., & O'Connor, M. E. (2003). Understanding persistence in bulimia nervosa: a 5-year naturalistic study. *Journal of Consulting and Clinical Psychology*, 71(1), 103–109. doi: 10.1037/0022-006X.71.1.103
- Fernandez-Aranda, F., Pinheiro, A. P., Tozzi, F., Via, M. L., Thornton, L. M., Plotnicov, K. H., ... Bulik, C. M. (2007). Symptom profile of major depressive disorder in women with eating disorders. *Australian & New Zealand Journal of Psychiatry*, 41(1), 24–31. doi: 10.1080/00048670601057718
- Fladung, A. K., Gron, G., Grammer, K., Herrnberger, B., Schilly, E., Grasteit, S., ... von Wietersheim, J. (2010). A neural signature of anorexia nervosa in the ventral striatal reward system. *American Journal of Psychiatry*, 167(2), 206–212. doi: 10.1176/appi.ajp.2009.09010071
- Forouzanfar, M. H., Alexander, L., Anderson, H. R., Bachman, V. F., Biryukov, S., Brauer, M., ... Murray, C. J. (2015). Global, regional and national comparative risk assessment of 79 behavioural, environmental and occupational and metabolic risks or clusters of risks in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *The Lancet*, 386(10010), 2287–2323. doi: 10.1016/S0140-6736(15)00128-2
- Frank, G. K., Kaye, W. H., Meltzer, C. C., Price, J. C., Greer, P., McConaha, C., & Skovira, K. (2002). Reduced 5-HT<sub>2A</sub> receptor binding after recovery from anorexia nervosa. *Biological Psychiatry*, 52(9), 896–906. doi: 10.1016/S0006-3223(02)01378-1
- Franko, D. L., Keshaviah, A., Eddy, K. T., Krishna, M., Davis, M. C., Keel, P. K., & Herzog, D. B. (2013). A longitudinal investigation of mortality in anorexia nervosa and bulimia nervosa. *American Journal of Psychiatry*, 170(8), 917–925. doi: 10.1176/appi.ajp.2013.12070868
- Fredrickson, B. L., & Roberts, T.-A. (1997). Objectification theory: Toward understanding women's lived experiences and mental health risks. *Psychology of Women Quarterly*, 21(2), 173–206. doi: 10.1111/j.1471-6402.1997.tb00108.x
- Frieling, H., Romer, K. D., Scholz, S., Mittelbach, F., Wilhelm, J., De Zwaan, M., ... Bleich, S. (2010). Epigenetic dysregulation of dopaminergic genes in eating disorders. *International Journal of Eating Disorders*, 43(7), 577–583. doi: 10.1002/eat.20745
- Garner, D. M., Olmstead, M. P., & Polivy, J. (1983). Development and validation of a multidimensional eating disorder inventory for anorexia nervosa and bulimia. *International Journal of Eating Disorders*, 2(2), 15–34. doi: 10.1002/1098-108X(198321)2:2<15::AID-EAT2260020203>3.0.CO;2-6
- The GBD 2013 Obesity Collaboration, Ng, M., Fleming, T., Robinson, M., Thomson, B., Graetz, N., ... Gakidou, E. (2014). Global, regional and national prevalence of overweight and obesity in children and adults 1980–2013: A systematic analysis. *Lancet*, 384(9945), 766–781. doi: 10.1016/S0140-6736(14)60460-8
- Gendall, K. A., Bulik, C. M., Joyce, P. R., McIntosh, V. V., & Carter, F. A. (2000). Menstrual cycle irregularity in bulimia nervosa: Associated factors and changes with treatment. *Journal of Psychosomatic Research*, 49(6), 409–415. doi: 10.1016/S0022-3999(00)00188-4
- Grabe, S., Ward, L. M., & Hyde, J. S. (2008). The role of the media in body image concerns among women: A meta-analysis of experimental and correlational studies. *Psychological Bulletin*, 134(3), 460–476. doi: 10.1037/0033-2909.134.3.460
- Griffiths, S., Mond, J. M., Murray, S. B., & Touyz, S. (2014). Young peoples' stigmatizing attitudes and beliefs about anorexia nervosa and muscle dysmorphia. *International Journal of Eating Disorders*, 47(2), 189–195. doi: 10.1002/eat.22220
- Griffiths, S., Murray, S. B., & Touyz, S. (2013). Disordered eating and the muscular ideal. *Journal of Eating Disorders*, 1(1), 15. doi: 10.1186/2050-2974-1-15
- Grilo, C. M., Crosby, R. D., Wilson, G. T., & Masheb, R. M. (2012). 12-month follow-up of fluoxetine and cognitive behavioral therapy for binge eating disorder. *Journal of Consulting and Clinical Psychology*, 80(6), 1108–1113. doi: 10.1037/a0030061
- Grilo, C. M., & Masheb, R. M. (2000). Onset of dieting vs binge eating in outpatients with binge eating disorder. *International Journal of Obesity and Related Disorders*, 24(4), 404–409. doi: 10.1038/sj.ijo.0801171
- Gudzune, K. A., Doshi, R. S., Mehta, A. K., Chaudhry, Z. W., Jacobs, D. K., Vakil, R. M., ... Clark, J. M. (2015). Efficacy of commercial weight-loss programs: an updated systematic review. *Annals of Internal Medicine*, 162(7), 501–512. doi: 10.7326/m14-2238
- Haedt-Matt, A. A., & Keel, P. K. (2011). Revisiting the affect regulation model of binge eating: A meta-analysis of studies using ecological momentary assessment. *Psychological Bulletin*, 137(4), 660–681. doi: 10.1037/a0023660

- Hargreaves, D., & Tiggemann, M. (2003). Longer-term implications of responsiveness to 'thin-ideal' television: support for a cumulative hypothesis of body image disturbance? *European Eating Disorders Review*, 11(6), 465–477. doi: 10.1002/erv.509
- Harris, J. L., Bargh, J. A., & Brownell, K. D. (2009). Priming effects of television food advertising on eating behavior. *Health Psychology*, 28(4), 404–413. doi: 10.1037/a0014399
- Hay, P. (2013). A systematic review of evidence for psychological treatments in eating disorders: 2005–2012. *International Journal of Eating Disorders*, 46(5), 462–469. doi: 10.1002/eat.22103
- Hay, P., Chinn, D., Forbes, D., Madden, S., Newton, R., Sugden, L., ... Ward, W. (2014). Royal Australian and New Zealand College of Psychiatrists clinical practice guidelines for the treatment of eating disorders. *Australian and New Zealand Journal of Psychiatry*, 48(11), 977–1008. doi: 10.1177/0004867414555814
- Hay, P. J., & Carriage, C. (2012). Eating disorder features in Indigenous Aboriginal and Torres Strait Islander Australian Peoples. *BMC Public Health*, 12(1), 233. doi: 10.1186/1471-2458-12-233
- Hay, P. J., Claudino, A. M., Touyz, S., & Abd Elbaky, G. (2015). Individual psychological therapy in the outpatient treatment of adults with anorexia nervosa. *Cochrane Database of Systematic Reviews*(7), CD003909. doi: 10.1002/14651858.CD003909.pub2
- Hay, P. P. J., Bacaltchuk, J., Stefano, S., & Kashyap, P. (2009). Psychological treatments for bulimia nervosa and bingeing. *Cochrane Database of Systematic Reviews*(4). doi: 10.1002/14651858.CD000562.pub3
- Healy, G. N., Wijndaele, K., Dunstan, D. W., Shaw, J. E., Salmon, J., Zimmet, P. Z., & Owen, N. (2008). Objectively measured sedentary time, physical activity and metabolic risk: the Australian Diabetes, Obesity and Lifestyle Study (AusDiab). *Diabetes Care*, 31(2), 369–371. doi: 10.2337/dc07-1795
- Herman, C. P., & Polivy, J. (1980). Restrained eating. In A. Stunkard (Ed.), *Obesity* (pp. 208–225). Philadelphia, PA: Saunders
- Herman, C. P., Polivy, J., Lank, C. N., & Heatherton, T. F. (1987). Anxiety, hunger and eating behavior. *Journal of Abnormal Psychology*, 96(3), 264–269. doi: 10.1037/0021-843X.96.3.264
- Herzog, D. B., Greenwood, D. N., Dorner, D. J., Flores, A. T., Ekeblad, E. R., Richards, A., ... Keller, M. B. (2000). Mortality in eating disorders: a descriptive study. *International Journal of Eating Disorders*, 28(1), 20–26. doi: 10.1002/(SICI)1098-108X(200007)28:1<20::AID-EAT3>3.0.CO;2-X.
- Hilbert, A., Bishop, M. E., Stein, R. I., Tanofsky-Kraff, M., Swenson, A. K., Welch, R. R., & Wilfley, D. E. (2012). Long-term efficacy of psychological treatments for binge eating disorder. *The British Journal of Psychiatry*, 200(3), 232–237. doi: 10.1192/bjp.bp.110.089664
- Hill, R., Haslett, C., & Kumar, S. (2001). Anorexia nervosa in an elderly woman. *Australian and New Zealand Journal of Psychiatry*, 35(2), 246–248.
- Ho, T. F., Tai, B. C., Lee, E. L., Cheng, S., & Liow, P. H. (2006). Prevalence and profile of females at risk of eating disorders in Singapore. *Singapore Medical Journal*, 47(6), 499–503.
- Hotta, M., Horikawa, R., Mabe, H., Yokoyama, S., Sugiyama, E., Yonekawa, T., ... Ogawa, Y. (2015). Epidemiology of anorexia nervosa in Japanese adolescents. *BioPsychoSocial Medicine*, 9(1), 17. doi: 10.1186/s13030-015-0044-2
- Hudson, J. I., Hiripi, E., Pope Jr, H. G., & Kessler, R. C. (2007). The prevalence and correlates of eating disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, 61(3), 348–358. doi: 10.1016/j.biopsych.2006.03.040
- Hudson, J. I., Lalonde, J. K., Berry, J. M., Pindyck, L. J., Bulik, C. M., Crow, S. J., ... Pope, H. G., Jr. (2006). Binge-eating disorder as a distinct familial phenotype in obese individuals. *Archives of General Psychiatry*, 63(3), 313–319. doi: 10.1001/archpsyc.63.3.313
- Hughes, E. K. (2012). Comorbid depression and anxiety in childhood and adolescent anorexia nervosa: Prevalence and implications for outcome. *Clinical Psychologist*, 16(1), 15–24. doi: 10.1111/j.1742-9552.2011.00034.x
- Jennings, P. S., Forbes, D., McDermott, B., Juniper, S., & Hulse, G. (2005). Acculturation and eating disorders in Asian and Caucasian Australian adolescent girls. *Psychiatry and Clinical Neurosciences*, 59(1), 56–61. doi: 10.1111/j.1440-1819.2005.01332.x
- Jett, S., LaPorte, D. J., & Wanchisn, J. (2010). Impact of exposure to pro-eating disorder websites on eating behaviour in college women. *European Eating Disorders Review*, 18(5), 410–416. doi: 10.1002/erv.1009
- Jiang, W., Tan, J., & Fassnacht, D. B. (2016). Implicit and explicit anti-fat bias among Asian females. *Eating and Weight Disorders — Studies on Anorexia, Bulimia and Obesity*, 1–9. doi: 10.1007/s40519-016-0290-8
- Johnson, J. G., Cohen, P., Kasen, S., & Brook, J. S. (2002). Childhood adversities associated with risk for eating disorders or weight problems during adolescence or early adulthood. *American Journal of Psychiatry*, 159(3), 394–400. doi: 10.1176/appi.ajp.159.3.394
- Josh, G., Korda, R. J., Attia, J., Liu, B., Bauman, A. E., & Banks, E. (2014). Body mass index and incident hospitalisation for cardiovascular disease in 158 546 participants from the 45 and Up Study. *International Journal of Obesity*, 38(6), 848–856. doi: 10.1038/ijo.2013.192
- Kang, J. H., Jeong, B. G., Cho, Y. G., Song, H. R., & Kim, K. A. (2011). Socioeconomic costs of overweight and obesity in Korean adults. *Journal of Korean Medical Science*, 26(12), 1533–1540.
- Kaye, W. (2008). Neurobiology of anorexia and bulimia nervosa Purdue Ingestive Behavior Research Center Symposium influences on eating and body weight over the lifespan: Children and adolescents. *Physiology & Behavior*, 94(1), 121–135. doi: 10.1016/j.physbeh.2007.11.037

- Kaye, W. H., Frank, G. K., Bailer, U. F., & Henry, S. E. (2005). Neurobiology of anorexia nervosa: Clinical implications of alterations of the function of serotonin and other neuronal systems. *International Journal of Eating Disorders*, 37(S1), S15–S19. doi: 10.1002/eat.20109
- Kaye, W. H., Frank, G. K., Meltzer, C. C., Price, J. C., McConaha, C. W., Crossan, P. J., . . . Rhodes, L. (2001). Altered serotonin 2A receptor activity in women who have recovered from bulimia nervosa. *American Journal of Psychiatry*, 158(7), 1152–1155. doi: 10.1176/appi.ajp.158.7.1152
- Keel, P. K., Baxter, M. G., Heatherton, T. F., & Joiner, T. E., Jr. (2007). A 20-year longitudinal study of body weight, dieting and eating disorder symptoms. *Journal of Abnormal Psychology*, 116(2), 422–432. doi: 10.1037/0021-843x.116.2.422
- Keel, P. K., & Brown, T. A. (2010). Update on course and outcome in eating disorders. *International Journal of Eating Disorders*, 43(3), 195–204. doi: 10.1002/eat.20810
- Keel, P. K., Mitchell, J. E., Davis, T. L., & Crow, S. J. (2002). Long-term impact of treatment in women diagnosed with bulimia nervosa. *International Journal of Eating Disorders*, 31(2), 151–158. doi: 10.1002/eat.10017
- Kenardy, J., Arnow, B., & Agras, W. S. (1996). The aversiveness of specific emotional states associated with binge-eating in obese subjects. *Australian and New Zealand Journal of Psychiatry*, 30(6), 839–844. doi: 10.3109/00048679609065053
- Kenardy, J., Brown, W. J., & Vogt, E. (2001). Dieting and health in young Australian women. *European Eating Disorders Review*, 9(4), 242–254. doi: 10.1002/erv.388
- Kessler, R. C., Berglund, P. A., Chiu, W. T., Deitz, A. C., Hudson, J. I., Shahly, V., . . . Xavier, M. (2013). The prevalence and correlates of binge eating disorder in the WHO World Mental Health Surveys. *Biological Psychiatry*, 73(9), 904–914. doi: 10.1016/j.biopsych.2012.11.020
- Keys, A., Brozek, J., Henschel, A., Mickelsen, O., & Taylor, H. L. (1950). *The Biology of Human Starvation (Vol. I-II)*. Minneapolis, MN: University of Minnesota Press.
- Klump, K. L., Wonderlich, S., Lehoux, P., Lilienfeld, L. R. R., & Bulik, C. (2002). Does environment matter? A review of nonshared environment and eating disorders. *International Journal of Eating Disorders*, 31(2), 118–135. doi: 10.1002/eat.10024
- Kopelman, P. G., & Caterson, I. D. (2009). Management of adult obesity. In P. G. Kopelman, I. D. Caterson & W. H. Dietz (Eds.), *Clinical Obesity in Adults and Children* (pp. 267–274). Oxford, UK: Wiley-Blackwell
- Kunkel, D., Wilcox, B., Cantor, J., Palmer, E., Linn, S., & Dowrick, P. (2004). *Report of the APA Task Force on Advertising and Children*. DC: American Psychological Association.
- Lal, A., Moodie, M., Ashton, T., Siahpush, M., & Swinburn, B. (2012). Health care and lost productivity costs of overweight and obesity in New Zealand. *Australian and New Zealand Journal of Public Health*, 36(6), 550–556. doi: 10.1111/j.1753-6405.2012.00931.x
- Lattimore, P. J., Wagner, H. L., & Gowers, S. (2000). Conflict avoidance in anorexia nervosa: an observational study of mothers and daughters. *European Eating Disorders Review*, 8(5), 355–368. doi: 10.1002/1099-0968(200010)8:5<355::AID-ERV368>3.0.CO;2-B
- Le, L. K.-D., Barendregt, J. J., Hay, P., & Mihalopoulos, C. (2017). Prevention of eating disorders: A systematic review and meta-analysis. *Clinical Psychology Review*, 53, April, 46–58. <http://dx.doi.org/10.1016/j.cpr.2017.02.001>.
- Le Grange, D. (2005). The Maudsley family-based treatment for adolescent anorexia nervosa. *World Psychiatry*, 4(3), 142–146.
- Le Grange, D., Crosby, R. D., Rathouz, P. J., & Leventhal, B. L. (2007). A randomized controlled comparison of family-based treatment and supportive psychotherapy for adolescent bulimia nervosa. *Archives of General Psychiatry*, 64(9), 1049–1056. doi: 10.1001/archpsyc.64.9.1049
- Le Grange, D., Lock, J., Loeb, K., & Nicholls, D. (2010). Academy for eating disorders position paper: The role of the family in eating disorders. *International Journal of Eating Disorders*, 43(1), 1–5. doi: 10.1002/eat.20751
- Lee, S. (1991). Anorexia nervosa in Hong Kong: a Chinese perspective. *Psychological Medicine*, 21(3), 703–711.
- Lee, S., Lee, A. M., Ngai, E., Lee, D. T., & Wing, Y. K. (2001). Rationales for food refusal in Chinese patients with anorexia nervosa. *International Journal of Eating Disorders*, 29(2), 224–229.
- Lee, S., Ng, K. L., Kwok, K., & Fung, C. (2010). The changing profile of eating disorders at a tertiary psychiatric clinic in Hong Kong (1987–2007). *International Journal of Eating Disorders*, 43(4), 307–314. doi: 10.1002/eat.20686
- Leit, R. A., Pope, H. G., & Gray, J. J. (2001). Cultural expectations of muscularity in men: The evolution of playgirl centerfolds. *International Journal of Eating Disorders*, 29(1), 90–93. doi: 10.1002/1098-108X(200101)29:1<90::AID-EAT15>3.0.CO;2-F
- Lock, J., & Le Grange, D. (2005). Family-based treatment of eating disorders. *International Journal of Eating Disorders*, 37(S1), S64–S67. doi: 10.1002/eat.20122
- Lock, J., Le Grange, D., Agras, W. S., Moye, A., Bryson, S. W., & Jo, B. (2010). Randomized clinical trial comparing family-based treatment with adolescent-focused individual therapy for adolescents with anorexia nervosa. *Archives of General Psychiatry*, 67(10), 1025–1032. doi: 10.1001/archgenpsychiatry.2010.128
- Ludwig, D. S., & Currie, J. (2010). The association between pregnancy weight gain and birthweight: a within-family comparison. *Lancet*, 376(9745), 984–990. doi: 10.1016/s0140-6736(10)60751-9
- Lundgren, J. D., Danoff-Burg, S., & Anderson, D. A. (2004). Cognitive-behavioral therapy for bulimia nervosa: An empirical analysis of clinical significance. *International Journal of Eating Disorders*, 35(3), 262–274. doi: 10.1002/eat.10254

- Marrazzi, M. A., Mullings-Britton, J., Stack, L., Powers, R. J., Lawhorn, J., Graham, V., ... Gunter, S. (1990). Atypical endogenous opioid systems in mice in relation to an auto-addiction opioid model of anorexia nervosa. *Life Sciences*, 47(16), 1427–1435.
- McCabe, M. P., Ricciardelli, L., Mellor, D., & Ball, K. (2005). Media influences on body image and disordered eating among Indigenous adolescent Australians. *Adolescence*, 40(157), 115–127.
- McCabe, R. E., McFarlane, T., Polivy, J., & Olmsted, M. P. (2001). Eating disorders, dieting and the accuracy of self-reported weight. *International Journal of Eating Disorders*, 29(1), 59–64.
- McFarlane, T., Polivy, J., & Herman, C. P. (1998). Effects of false weight feedback on mood, self-evaluation and food intake in restrained and unrestrained eaters. *Journal of Abnormal Psychology*, 107(2), 312–318.
- Mehler, P. S., Krantz, M. J., & Sachs, K. V. (2015). Treatments of medical complications of anorexia nervosa and bulimia nervosa. *Journal of Eating Disorders*, 3(1), 15. doi: 10.1186/s40337-015-0041-7
- Melioli, T., Bauer, S., Franko, D. L., Moessner, M., Ozer, F., Chabrol, H., & Rodgers, R. F. (2016). Reducing eating disorder symptoms and risk factors using the internet: A meta-analytic review. *International Journal of Eating Disorders*, 49(1), 19–31. doi: 10.1002/eat.22477
- Micali, N., Martini, M. G., Thomas, J. J., Eddy, K. T., Kothari, R., Russell, E., ... Treasure, J. (2017). Lifetime and 12-month prevalence of eating disorders amongst women in mid-life: a population-based study of diagnoses and risk factors. *BMC Medicine*, 15, 12. <http://doi.org/10.1186/s12916-016-0766-4>
- Moodie, A. R., Tolhurst, P., & Martin, J. E. (2016). Australia's health: being accountable for prevention. *Medical Journal of Australia*, 204(6), 223–225.
- Mulgrew, K. E., & Cragg, D. N. (2015). Age differences in body image responses to idealized male figures in music television. *Journal of Health Psychology*, 1359105315616177. doi: 10.1177/1359105315616177
- Murray, S. B., Griffiths, S., & Mond, J. M. (2016). Evolving eating disorder psychopathology: conceptualising muscularity-oriented disordered eating. *The British Journal of Psychiatry*, 208(5), 414–415. doi: 10.1192/bjp.bp.115.168427
- Murray, S. B., Rieger, E., Hildebrandt, T., Karlov, L., Russell, J., Boon, E., ... Touyz, S. W. (2012). A comparison of eating, exercise, shape and weight related symptomatology in males with muscle dysmorphia and anorexia nervosa. *Body Image*, 9(2), 193–200. doi: 10.1016/j.bodyim.2012.01.008
- National Health and Medical Research Council. (2013). *Australian Dietary Guidelines*. Canberra: NHMRC.
- Neumark-Sztainer, D., Paxton, S. J., Hannan, P. J., Haines, J., & Story, M. (2006). Does body satisfaction matter? Five-year longitudinal associations between body satisfaction and health behaviors in adolescent females and males. *Journal of Adolescent Health*, 39(2), 244–251. doi: 10.1016/j.jadohealth.2005.12.001
- Nicholls, D. E., & Viner, R. M. (2009). Childhood risk factors for lifetime anorexia nervosa by age 30 years in a national birth cohort. *Journal of the American Academy of Child & Adolescent Psychiatry*, 48(8), 791–799. doi: 10.1097/CHI.0b013e3181ab8b75
- Norman, R. E., Byambaa, M., De, R., Butchart, A., Scott, J., & Vos, T. (2012). The long-term health consequences of child physical abuse, emotional abuse and neglect: A systematic review and meta-analysis. *PLOS Medicine*, 9(11), e1001349. doi: 10.1371/journal.pmed.1001349
- Obesity Working Group Australia. (2009). *Australia: the healthiest country by 2020*. Canberra: OWGA.
- Patton, G. C., Selzer, R., Coffey, C., Carlin, J. B., & Wolfe, R. (1999). Onset of adolescent eating disorders: population based cohort study over 3 years. *BMJ*, 318(7186), 765–768. doi: 10.1136/bmj.318.7186.765
- Paxton, S. J., McLean, S. A., Gollings, E. K., Faulkner, C., & Wertheim, E. H. (2007). Comparison of face-to-face and internet interventions for body image and eating problems in adult women: An RCT. *International Journal of Eating Disorders*, 40(8), 692–704. doi: 10.1002/eat.20446
- Paxton, S. J., Wertheim, E. H., Gibbons, K., Szumukler, G. I., Hillier, L., & Petrovich, J. L. (1991). Body image satisfaction, dieting beliefs and weight loss behaviors in adolescent girls and boys. *Journal of Youth and Adolescence*, 20(3), 361–379. doi: 10.1007/bf01537402
- Peat, C., Mitchell, J. E., Hoek, H., & Wonderlich, S. (2009). Validity and utility of subtyping anorexia nervosa. *International Journal of Eating Disorders*, 42(7), 590–594. doi: 10.1002/eat.20717
- Pike, K. M., & Dunne, P. E. (2015). The rise of eating disorders in Asia: a review. *Journal of Eating Disorders*, 3, 33. doi: 10.1186/s40337-015-0070-2
- Pitayatananan, P., Butchon, R., Yothasamut, J., Aekplakorn, W., Teerawattananon, Y., Suksomboon, N., & Thavorncharoensap, M. (2014). Economic costs of obesity in Thailand: a retrospective cost-of-illness study. *BMC Health Services Research*, 14(1), 146. doi: 10.1186/1472-6963-14-146
- Polivy, J. (1976). Perception of calories and regulation of intake in restrained and unrestrained subjects. *Addictive Behaviors*, 1(3), 237–243. doi: 10.1016/0306-4603(76)90016-2
- Polivy, J., Heatherton, T. F., & Herman, C. P. (1988). Self-esteem, restraint and eating behavior. *Journal of Abnormal Psychology*, 97(3), 354–356.
- Polivy, J., & Herman, C. P. (1985). Dieting and bingeing. A causal analysis. *The American Psychologist*, 40(2), 193–201.
- Polivy, J., Herman, C. P., & Howard, K. (1988). The Restraint Scale: Assessment of dieting. In M. Hersen & A. S. Bellack (Eds.), *Dictionary of behavioral assessment techniques* (pp. 377–380). Elmsford, NY: Pergamon Press.
- Polivy, J., Herman, C. P., & McFarlane, T. (1994). Effects of anxiety on eating: does palatability moderate distress-induced overeating in dieters? *Journal of Abnormal Psychology*, 103(3), 505–510.

- Pope, H. G., Jr., Lalonde, J. K., Pindyck, L. J., Walsh, T., Bulik, C. M., Crow, S. J., . . . Hudson, J. I. (2006). Binge eating disorder: a stable syndrome. *American Journal of Psychiatry*, 163(12), 2181–2183. doi: 10.1176/ajp.2006.163.12.2181
- Poulsen, S., Lunn, S., Daniel, S. I. F., Folke, S., Mathiesen, B. B., Katznelson, H., & Fairburn, C. G. (2014). A randomized controlled trial of psychoanalytic psychotherapy or cognitive-behavioral therapy for bulimia nervosa. *American Journal of Psychiatry*, 171(1), 109–116. doi: 10.1176/appi.ajp.2013.12121511
- Presnell, K., Stice, E., Seidel, A., & Madeley, M. C. (2009). Depression and eating pathology: Prospective reciprocal relations in adolescents. *Clinical Psychology & Psychotherapy*, 16(4), 357–365. doi: 10.1002/cpp.630
- Puhl, R. M., & Heuer, C. A. (2009). The stigma of obesity: A review and update. *Obesity*, 17(5), 941–964. doi: 10.1038/oby.2008.636
- Ranzenhofer, L. M., Engel, S. G., Crosby, R. D., Anderson, M., Vannucci, A., Cohen, L. A., . . . Tanofsky-Kraff, M. (2014). Using ecological momentary assessment to examine interpersonal and affective predictors of loss of control eating in adolescent girls. *International Journal of Eating Disorders*, 47(7), 748–757. doi: 10.1002/eat.22333
- Reas, D. L., & Grilo, C. M. (2015). Pharmacological treatment of binge eating disorder: update review and synthesis. *Expert Opinion on Pharmacotherapy*, 16(10), 1463–1478. doi: 10.1517/14656566.2015.1053465
- Reba-Harrelson, L., Von Holle, A., Hamer, R. M., Swann, R., Reyes, M. L., & Bulik, C. M. (2009). Patterns and prevalence of disordered eating and weight control behaviors in women ages 25–45. *Eating and Weight Disorders*, 14(4), e190–198.
- Reidpath, D. D., Burns, C., Garrard, J., Mahoney, M., & Townsend, M. (2002). An ecological study of the relationship between social and environmental determinants of obesity. *Health & Place*, 8(2), 141–145. doi: 10.1016/S1353-8292(01)00028-4
- Ricciardelli, L. A., & McCabe, M. P. (2001). Dietary restraint and negative affect as mediators of body dissatisfaction and bulimic behavior in adolescent girls and boys. *Behaviour Research and Therapy*, 39(11), 1317–1328. doi: 10.1016/S0005-7967(00)00097-8
- Rieger, E., Wilfley, D. E., Stein, R. I., Marino, V., & Crow, S. J. (2005). A comparison of quality of life in obese individuals with and without binge eating disorder. *International Journal of Eating Disorders*, 37(3), 234–240. doi: 10.1002/eat.20101
- Rodgers, R. F., & DuBois, R. H. (2016). Cognitive biases to appearance-related stimuli in body dissatisfaction: A systematic review. *Clinical Psychology Review*, 46, 1–11. doi: 10.1016/j.cpr.2016.04.006
- Rodgers, R. F., Paxton, S. J., & Chabrol, H. (2009). Effects of parental comments on body dissatisfaction and eating disturbance in young adults: A sociocultural model. *Body Image*, 6(3), 171–177. doi: 10.1016/j.bodyim.2009.04.004
- Rouleau, C. R., & von Ranson, K. M. (2011). Potential risks of pro-eating disorder websites. *Clinical Psychology Review*, 31(4), 525–531. doi: 10.1016/j.cpr.2010.12.005
- Sacks, F. M., Bray, G. A., Carey, V. J., Smith, S. R., Ryan, D. H., Anton, S. D., . . . Williamson, D. A. (2009). Comparison of weight-loss diets with different compositions of fat, protein and carbohydrates. *New England Journal of Medicine*, 360(9), 859–873. doi: 10.1056/NEJMoa0804748
- Sacks, G., Swinburn, B. A., & Lawrence, M. A. (2008). A systematic policy approach to changing the food system and physical activity environments to prevent obesity. *Australia and New Zealand Health Policy*, 5(1), 13. doi: 10.1186/1743-8462-5-13
- Seifert, T. (2005). Anthropomorphic characteristics of centerfold models: Trends towards slender figures over time. *International Journal of Eating Disorders*, 37(3), 271–274. doi: 10.1002/eat.20086
- Selby, E. A., Cornelius, T., Fehling, K. B., Kranzler, A., Panza, E. A., Lavender, J. M., . . . Le Grange, D. (2015). A perfect storm: examining the synergistic effects of negative and positive emotional instability on promoting weight loss activities in anorexia nervosa. *Frontiers in Psychology*, 6, 1260. doi: 10.3389/fpsyg.2015.01260
- Selby, E. A., Wonderlich, S. A., Crosby, R. D., Engel, S. G., Panza, E., Mitchell, J. E., . . . Grange, D. L. (2014). Nothing tastes as good as thin feels. *Clinical Psychological Science*, 2(4), 514–531. doi: 10.1177/2167702613512794
- Silventoinen, K., Rokholm, B., Kaprio, J., & Sorensen, T. I. (2010). The genetic and environmental influences on childhood obesity: a systematic review of twin and adoption studies. *International Journal of Obesity*, 34(1), 29–40. doi: 10.1038/ijo.2009.177
- Slater, A., & Tiggemann, M. (2010). Body image and disordered eating in adolescent girls and boys: A test of objectification theory. *Sex Roles*, 63(1), 42–49. doi: 10.1007/s11199-010-9794-2
- Slevec, J. H., & Tiggemann, M. (2011). Predictors of body dissatisfaction and disordered eating in middle-aged women. *Clinical Psychology Review*, 31(4), 515–524. doi: 10.1016/j.cpr.2010.12.002
- Smink, F. R. E., van Hoeken, D., & Hoek, H. W. (2012). Epidemiology of eating disorders: Incidence, prevalence and mortality rates. *Current Psychiatry Reports*, 14(4), 406–414. doi: 10.1007/s11920-012-0282-y
- Smith, D. G., & Robbins, T. W. (2013). The neurobiological underpinnings of obesity and binge eating: a rationale for adopting the food addiction model. *Biological Psychiatry*, 73(9), 804–810. doi: 10.1016/j.biopsych.2012.08.026
- Smith, E., & Rieger, E. (2006). The effect of attentional bias toward shape- and weight-related information on body dissatisfaction. *International Journal of Eating Disorders*, 39(6), 509–515. doi: 10.1002/eat.20291
- Smithers, L. G., Lynch, J. W., & Merlin, T. (2014). Industry self-regulation and TV advertising of foods to Australian children. *Journal of Paediatrics and Child Health*, 50(5), 386–392. doi: 10.1111/jpc.12488
- Smyth, J. M., Wonderlich, S. A., Heron, K. E., Sliwinski, M. J., Crosby, R. D., Mitchell, J. E., & Engel, S. G. (2007). Daily and momentary mood and stress are associated with binge eating and vomiting in bulimia nervosa patients in the natural environment. *Journal of Consulting and Clinical Psychology*, 75(4), 629–638. doi: 10.1037/0022-006x.75.4.629

- Spataro, J., Mullen, P. E., Burgess, P. M., Wells, D. L., & Moss, S. A. (2004). Impact of child sexual abuse on mental health. *Prospective Study in Males and Females*, 184(5), 416–421. doi: 10.1192/bjp.184.5.416
- Steele, A. L., Bergin, J., & Wade, T. D. (2011). Self-efficacy as a robust predictor of outcome in guided self-help treatment for broadly defined bulimia nervosa. *International Journal of Eating Disorders*, 44(5), 389–396. doi: 10.1002/eat.20830
- Steinhausen, H. (2002). The outcome of anorexia nervosa in the 20th Century. *American Journal of Psychiatry*, 159(8), 1284–1293. doi: 10.1176/appi.ajp.159.8.1284
- Steinhausen, H. C., & Weber, S. (2009). The outcome of bulimia nervosa: findings from one-quarter century of research. *American Journal of Psychiatry*, 166(12), 1331–1341. doi: 10.1176/appi.ajp.2009.09040582
- Stice, E. (2002). Risk and maintenance factors for eating pathology: a meta-analytic review. *Psychological Bulletin*, 128(5), 825–848.
- Stice, E., Marti, C. N., Spoor, S., Presnell, K., & Shaw, H. (2008). Dissonance and healthy weight eating disorder prevention programs: long-term effects from a randomized efficacy trial. *Journal of Consulting and Clinical Psychology*, 76(2), 329–340. doi: 10.1037/0022-006x.76.2.329
- Stice, E., Shaw, H., & Marti, C. N. (2007). A meta-analytic review of eating disorder prevention programs: encouraging findings. *Annual Review of Clinical Psychology*, 3, 207–231. doi: 10.1146/annurev.clinpsy.3.022806.091447
- Stice, E., & Whitenton, K. (2002). Risk factors for body dissatisfaction in adolescent girls: a longitudinal investigation. *Developmental Psychology*, 38(5), 669–678.
- Støving, R. K., Hangaard, J., Hansen-Nord, M., & Hagen, C. (1999). A review of endocrine changes in anorexia nervosa. *Journal of Psychiatric Research*, 33(2), 139–152. doi: 10.1016/S0022-3956(98)00049-1
- Striegel-Moore, R. H., & Bulik, C. M. (2007). Risk factors for eating disorders. *The American Psychologist*, 62(3), 181–198. doi: 10.1037/0003-066x.62.3.181
- Striegel-Moore, R. H., Fairburn, C. G., Wilfley, D. E., Pike, K. M., Dohm, F. A., & Kraemer, H. C. (2005). Toward an understanding of risk factors for binge-eating disorder in black and white women: a community-based case-control study. *Psychological Medicine*, 35(6), 907–917.
- Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2000). Controlled family study of anorexia nervosa and bulimia nervosa: evidence of shared liability and transmission of partial syndromes. *American Journal of Psychiatry*, 157(3), 393–401. doi: 10.1176/appi.ajp.157.3.393
- Strober, M., Freeman, R., Lampert, C., Diamond, J., & Kaye, W. (2001). Males with anorexia nervosa: A controlled study of eating disorders in first-degree relatives. *International Journal of Eating Disorders*, 29(3), 263–269. doi: 10.1002/eat.1017
- Strober, M., Freeman, R., Lampert, C., Diamond, J., Teplinsky, C., & DeAntonio, M. (2006). Are there gender differences in core symptoms, temperament and short-term prospective outcome in anorexia nervosa? *International Journal of Eating Disorders*, 39(7), 570–575. doi: 10.1002/eat.20293
- Swami, V., Frederick, D. A., Aavik, T., Alcalay, L., Allik, J., Anderson, D., ... Zivcic-Becirevic, I. (2010). The attractive female body weight and female body dissatisfaction in 26 countries across 10 world regions: results of the international body project I. *Personality & Social Psychology Bulletin*, 36(3), 309–325. doi: 10.1177/0146167209359702
- Swinbourne, J., Hunt, C., Abbott, M., Russell, J., Clare, T. S., & Touyz, S. (2012). The comorbidity between eating disorders and anxiety disorders: Prevalence in an eating disorder sample and anxiety disorder sample. *Australian & New Zealand Journal of Psychiatry*, 46(2), 118–131. doi: 10.1177/0004867411432071
- Swinburn, B., Kraak, V., Rutter, H., Vandevijvere, S., Lobstein, T., Sacks, G., ... Magnusson, R. (2015). Strengthening of accountability systems to create healthy food environments and reduce global obesity. *Lancet*, 385(9986), 2534–2545. doi: 10.1016/S0140-6736(14)61747-5
- Thompson, J. K., & Stice, E. (2001). Thin-ideal internalization: Mounting evidence for a new risk factor for body-image disturbance and eating pathology. *Current Directions in Psychological Science*, 10(5), 181–183. doi: 10.1111/1467-8721.00144
- Tiggemann, M. (2013). Objectification Theory: Of relevance for eating disorder researchers and clinicians? *Clinical Psychologist*, 17(2), 35–45. doi: 10.1111/cp.12010
- Tiggemann, M., & Slater, A. (2004). Thin ideals in music television: A source of social comparison and body dissatisfaction. *International Journal of Eating Disorders*, 35(1), 48–58. doi: 10.1002/eat.10214
- Tiggemann, M., & Slater, A. (2013). NetGirls: The internet, Facebook and body image concern in adolescent girls. *International Journal of Eating Disorders*, 46(6), 630–633. doi: 10.1002/eat.22141
- Timperio, A., Cameron-Smith, D., Burns, C., & Crawford, D. (2000). The public's response to the obesity epidemic in Australia: weight concerns and weight control practices of men and women. *Public Health Nutrition*, 3(4), 417–424.
- Tomiya, A. J., Finch, L. E., Belsky, A. C., Buss, J., Finley, C., Schwartz, M. B., & Daubenmier, J. (2015). Weight bias in 2001 versus 2013: contradictory attitudes among obesity researchers and health professionals. *Obesity (Silver Spring)*, 23(1), 46–53. doi: 10.1002/oby.20910
- Tong, J., Miao, S., Wang, J., Yang, F., Lai, H., Zhang, C., ... Hsu, L. K. (2014). A two-stage epidemiologic study on prevalence of eating disorders in female university students in Wuhan, China. *Social Psychiatry and Psychiatric Epidemiology*, 49(3), 499–505. doi: 10.1007/s00127-013-0694-y
- Touyz, S., Le Grange, D., Lacey, H., Hay, P., Smith, R., Maguire, S., ... Crosby, R. D. (2013). Treating severe and enduring anorexia nervosa: a randomized controlled trial. *Psychological Medicine*, 43(12), 2501–2511. doi: 10.1017/s0033291713000949

- Treasure, J., & Schmidt, U. (2013). The cognitive-interpersonal maintenance model of anorexia nervosa revisited: a summary of the evidence for cognitive, socio-emotional and interpersonal predisposing and perpetuating factors. *Journal of Eating Disorders*, 1(1), 13. doi: 10.1186/2050-2974-1-13
- Vocks, S., Tuschen-Caffier, B., Pietrowsky, R., Rustenbach, S. J., Kersting, A., & Herpertz, S. (2010). Meta-analysis of the effectiveness of psychological and pharmacological treatments for binge eating disorder. *International Journal of Eating Disorders*, 43(3), 205–217. doi: 10.1002/eat.20696
- Vohs, K. D., & Heatherton, T. F. (2000). Self-regulatory failure: a resource-depletion approach. *Psychological Science*, 11(3), 249–254.
- Volkow, N. D., Wang, G. J., Fowler, J. S., Logan, J., Jayne, M., Franceschi, D., . . . Pappas, N. (2002). 'Nonhedonic' food motivation in humans involves dopamine in the dorsal striatum and methylphenidate amplifies this effect. *Synapse*, 44(3), 175–180. doi: 10.1002/syn.10075
- Wade, T. D., Bergin, J. L., Tiggemann, M., Bulik, C. M., & Fairburn, C. G. (2006). Prevalence and long-term course of lifetime eating disorders in an adult Australian twin cohort. *Australian & New Zealand Journal of Psychiatry*, 40(2), 121–128. doi: 10.1080/j.1440-1614.2006.01758.x
- Wade, T. D., Bulik, C. M., Neale, M., & Kendler, K. S. (2000). Anorexia nervosa and major depression: Shared genetic and environmental risk factors. *American Journal of Psychiatry*, 157(3), 469–471. doi: 10.1176/appi.ajp.157.3.469
- Wade, T. D., Treloar, S. A., Heath, A. C., & Martin, N. G. (2009). An examination of the overlap between genetic and environmental risk factors for intentional weight loss and overeating. *International Journal of Eating Disorders*, 42(6), 492–497. doi: 10.1002/eat.20668
- Wang, Z., Hoy, W. E., & Si, D. (2010). Incidence of type 2 diabetes in Aboriginal Australians: an 11-year prospective cohort study. *BMC Public Health*, 10, 487. doi: 10.1186/1471-2458-10-487
- Wansink, B., & Payne, C. R. (2009). The joy of cooking too much: 70 years of calorie increases in classic recipes. *Annals of Internal Medicine*, 150(4), 291–292.
- Wansink, B., & Sobal, J. (2007). Mindless eating. *Environment and Behavior*, 39(1), 106–123. doi:10.1177/0013916506295573
- Waters, A., Hill, A., & Waller, G. (2001). Internal and external antecedents of binge eating episodes in a group of women with bulimia nervosa. *International Journal of Eating Disorders*, 29(1), 17–22.
- WHO Expert Consultation. (2004). Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet*, 363(9403), 157–163. doi: 10.1016/s0140-6736(03)15268-3
- Wilson, G. T., & Sysko, R. (2009). Frequency of binge eating episodes in bulimia nervosa and binge eating disorder: Diagnostic considerations. *International Journal of Eating Disorders*, 42(7), 603–610. doi: 10.1002/eat.20726
- Winzelberg, A. J., Eppstein, D., Eldredge, K. L., Wilfley, D., Dasmahapatra, R., Dev, P., & Taylor, C. B. (2000). Effectiveness of an Internet-based program for reducing risk factors for eating disorders. *Journal of Consulting and Clinical Psychology*, 68(2), 346–350.
- Wolfe, B. E., Baker, C. W., Smith, A. T., & Kelly-Weeder, S. (2009). Validity and utility of the current definition of binge eating. *International Journal of Eating Disorders*, 42(8), 674–686. doi: 10.1002/eat.20728
- Wonderlich, S. A., Gordon, K. H., Mitchell, J. E., Crosby, R. D., & Engel, S. G. (2009). The validity and clinical utility of binge eating disorder. *International Journal of Eating Disorders*, 42(8), 687–705. doi: 10.1002/eat.20719
- Yilmaz, Z., Hardaway, J. A., & Bulik, C. M. (2015). Genetics and epigenetics of eating disorders. *Advances in Genomics and Genetics*, 5, 131–150. doi: 10.2147/AGG.S55776
- Zellner, D. A., Harner, D. E., & Adler, R. L. (1989). Effects of eating abnormalities and gender on perceptions of desirable body shape. *Journal of Abnormal Psychology*, 98(1), 93–96.
- Zhao, W., Zhai, Y., Hu, J., Wang, J., Yang, Z., Kong, L., & Chen, C. (2008). Economic burden of obesity-related chronic diseases in Mainland China. *Obesity Reviews*, 9, 62–67. doi: 10.1111/j.1467-789X.2007.00440.x
- Zipfel, S., Wild, B., Gross, G., Friederich, H. C., Teufel, M., Schellberg, D., . . . Herzog, W. (2014). Focal psychodynamic therapy, cognitive behaviour therapy and optimised treatment as usual in outpatients with anorexia nervosa (ANTOP study): randomised controlled trial. *Lancet*, 383(9912), 127–137. doi: 10.1016/s0140-6736(13)61746-8
- Zwicker, K., & Rieger, E. (2013). Stigmatizing attitudes towards individuals with anorexia nervosa: an investigation of attribution theory. *Journal of Eating Disorders*, 1(1), 5. doi: 10.1186/2050-2974-1-5

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## CHAPTER 9

# Sexual disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 9.1** describe the influence of culture and gender on sexual norms and summarise the sexual response cycle for men and women
  - 9.2** explain the symptoms, causes and treatments for sexual dysfunctions
  - 9.3** explain the symptoms, causes and treatments for paraphilic disorders.
-

## OPENING SCENARIO

My name is Chris. I was biologically born a female, raised by loving parents and two older sisters. I quickly realised that I was different to my sisters. I didn't want to dress like them or even do similar things to what they were doing. I was convinced from a very young age that I was simply born in the wrong body. If you asked me to explain it, that's all I could say — my physical body did not match my insides (thoughts and feelings). I didn't want breasts or a vagina. My penis and testicles were missing. I would tape down my breasts and avoid social situations; it was constantly on my mind. When I first discussed this with my parents, they brushed it aside and were convinced that I was a lesbian. They told me that it was 'okay' and that they would accept me as a homosexual. But that didn't help — I felt unheard, I felt like I was in prison, stuck in a body that didn't belong to me.

I finally decided to speak to a professional, someone who knew something — anything — about what I was going through, how to cope and how to manage. That was the first day of my new life! I wasn't 'confused' and there were actually steps I could take to feel 'normal in myself'. Finally, an opportunity for my insides and my physical body to align. I felt relieved that my name was 'Chris' — a gender-neutral name — I only now realise that this made things so much easier for my parents when I decided to proceed with transitioning from female to male. The decision to transition was really easy, it was the questions from friends and family that were difficult: 'Are you sure you want to go ahead with this?', 'Is this just a phase?', 'What if you change your mind?', 'Why don't you wait a few more years?' and 'Don't you want to have kids?' — all questions that made me feel as though I had to continually justify 'being me'.

Don't get me wrong, the process was long — but I've never been happier in my life.

### QUESTIONS

1. If you were using an outdated version of the DSM, such as the DSM-III to diagnose Chris, what could be a potential diagnosis?
2. Using the DSM-5, what diagnosis would Chris be potentially provided with? Justify your response.

## Introduction

Sexuality is one of the most personal areas of life. Each of us is a sexual being with preferences and fantasies that may surprise or even shock us from time to time. Usually, these are part of normal sexual functioning. But when our fantasies or desires begin to affect us or others in unwanted or harmful ways, they begin to qualify as abnormal.

For perspective, we begin by briefly describing norms and healthy sexual behaviour. Then we consider two forms of sexual problems: sexual dysfunctions and paraphilic disorders. The American Psychiatric Association (APA) defines sexual dysfunctions as a 'heterogeneous group of disorders that are typically characterised by a clinically significant disturbance in a person's ability to respond sexually or to experience sexual pleasure' (APA, 2013a, p. 423). Paraphilic disorders, on the other hand, are defined as 'a paraphilia that is currently causing distress or impairment to the individual or a paraphilia whose satisfaction has entailed personal harm or risk of harm, to others' (APA, 2013a, p. 685), where **paraphilia** refers to an intense sexual fantasy, urge or behaviour involving an object, suffering or humiliation, or non-consenting partner, which causes distress to the person experiencing it.

## 9.1 Sexual norms and behaviour

**LEARNING OUTCOME 9.1** Describe the influence of culture and gender on sexual norms and summarise the sexual response cycle for men and women.

What society defines as normal or desirable in human sexual behaviour varies with time and place. In contemporary Western worldviews, *inhibition* of sexual expression is seen as a problem. Contrast this with nineteenth- and early-twentieth-century views that *excess* was the culprit; in particular, excessive

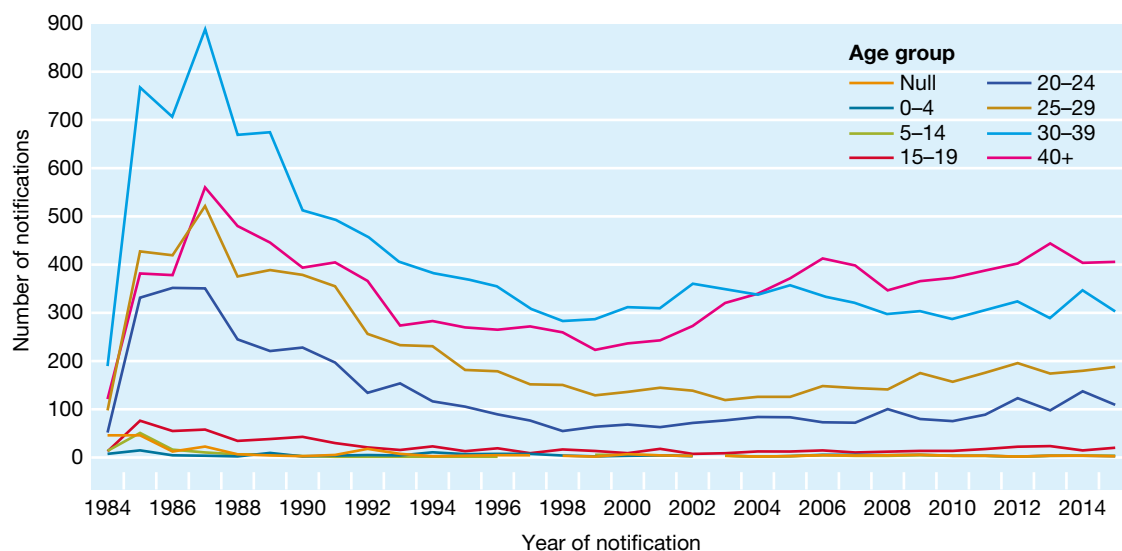
masturbation in childhood was widely believed to lead to sexual problems in adulthood. Von Krafft-Ebing (1902) proposed that early masturbation damaged the sexual organs and exhausted a finite reservoir of sexual energy, resulting in diminished ability to function sexually in adulthood. Even in adulthood, excessive sexual activity was thought to underlie problems such as erectile failure. The general Victorian view was that sexual appetite was dangerous and therefore had to be restrained. For example, to discourage children from handling their genitals, metal mittens were promoted; to distract adults from too much sex, outdoor exercise and a bland diet were recommended. In fact, Kellogg's Corn Flakes, developed by John Harvey Kellogg, were developed as foods that would decrease masturbation. Needless to say, they didn't!

Other historical changes have influenced people's attitudes and experiences of sexuality. For example, technology has changed sexual experiences, as the number of people accessing sexual content on the internet increased dramatically over the past decade. Even as the accessibility of sexual content increased dramatically, AIDS and other sexually transmitted diseases changed the risks associated with sexual behaviour. Rates of sexually transmitted infections (STIs) in Australia have increased, while HIV rates remain stable (Kirby Institute, 2016). In Australia, by the end of 2015, there were an estimated 25 313 people living with HIV and approximately 10 percent were unaware of their predicament (Australian Federation of AIDS Organisations, 2016). See figure 9.1 for recent HIV statistics. Other changes are influencing sexual norms as well. As the population of Australia ages one can hope that the emphasis on the right to a good sex life until the day one dies will emerge.

Norms about sexuality have fluctuated a great deal over time. In the early twentieth century, cornflakes were promoted as part of a bland diet designed to reduce masturbation.



**FIGURE 9.1** Number of HIV diagnoses in Australia by age group from 1984–2015



Source: Kirby Institute (2017).

Aside from changes over time and across generations, culture influences attitudes and beliefs about sexuality as well. In some cultures, sexuality is viewed as an important part of wellbeing and pleasure, whereas in others, sexuality is seen as relevant only for procreation (Bhugra, Popelyuk, & McMullen, 2010). Cultures also vary in their acceptance of variations in sexual behaviour. For example, among Sambians living in Papua New Guinea, Herdt (1999) wrote about rituals in which pubescent males engage in oral sex with older men as a way of learning about their sexuality. In other cultures it is common to stigmatise same-gender sexual behaviour. Clearly, we must keep varying cultural norms in mind as we study human sexual behaviour. See focus on discovery 9.1 for a look at the complicated path taken by health professionals in response to changing attitudes towards sexual orientation.

#### FOCUS ON DISCOVERY 9.1

##### Learning from history

There has been a long history of debate over the DSM's approach to sexual orientation (the preference for a male or female partner) and gender identity (the sense of the self as male or female). Gender identity is distinct from sexual orientation. Many have argued that the DSM's approach to both has fostered stigma.

Until 1973, homosexuality was listed in the DSM as one of the sexual disorders. In 1973, the Nomenclature Committee (now known as the Sexual and Gender Identity Disorders Work Group) of the American Psychiatric Association, under pressure from professional and activist groups, recommended the elimination of the category 'homosexuality' and the substitution of 'sexual orientation disturbance'. This new diagnosis was to be applied to gay men and women who are 'disturbed by, in conflict with or wish to change their sexual orientation'. The change was approved, but not without heated protests from several renowned psychiatrists who remained convinced that homosexuality reflects a fixation at an early stage of psychosexual development and is therefore inherently abnormal. Today, these protests would be considered misguided, prejudiced and antiscientific.

Pathologising homosexuality seems particularly odd when one considers how common such behaviour is. For example, in a study that involved interviews with 20 094 participants, approximately 9 percent of men and 19 percent of women report a history of same-sex attraction and/or experience (Richters et al., 2014).

In the 1980 publication of the DSM-III, the Nomenclature Committee dithered by adding a new category called *ego-dystonic homosexuality*, which referred to a person who is homosexually aroused, is persistently distressed by this arousal and wishes to become heterosexual. This category was dropped in later editions of the DSM. The DSM-IV and the DSM-IV-TR include the catchall category of 'sexual disorder not otherwise specified', which refers to 'persistent and marked distress about one's sexual orientation', which can be applied when a person is distressed over a heterosexual or homosexual orientation. The logic of this diagnosis is flawed. At the same time that the DSM had ceased labelling homosexuality as abnormal, a gay man or lesbian is considered abnormal if he or she has been persuaded by a prejudiced society that his or her sexual orientation is disordered.

Catherine 'Cate' McGregor, formerly Malcolm Gerard McGregor, was born biologically male and is a serving member of the Australian Defence Force. When asked about gender reassignment surgery, Cate was quoted as saying 'it's about being congruent in your identity, I would like to feel whole' (McGregor, 2015).



Although homosexuality is no longer diagnosed, **gender dysphoria** remains in the DSM-5 and the diagnosis provokes similar controversy. Some people feel deep within themselves, usually from early childhood, that they are of the opposite sex. They are not persuaded by the presence of their genitals, nor by others' perceptions of their gender. A man can look at himself in a mirror, see the body of a biological man and yet experience that body as belonging to a woman. He may want to surgically alter his body to bring it in line with his gender identity. The DSM-5 includes a diagnosis of gender dysphoria for people who experience a strong and persistent identification with the opposite sex. Parallel with the 1980 DSM criteria for ego-dystonic homosexuality, gender dysphoria is only diagnosed when the desire to be a member of the opposite sex causes marked distress or functional impairment. Many would argue that the distress reflects an internalisation of the stigma faced by people who violate gender roles.

Gender dysphoria is one of the most debated categories in the DSM (Vance et al., 2010). The DSM-5 has now acknowledged that there are people with the intense belief that they are a member of the opposite sex by replacing the diagnostic name of 'gender identity disorder' with 'gender dysphoria'. It is no longer labelled as a disorder and the critical element is the presence of clinically significant distress associated with the condition (APA, 2013b).

Take a moment to consider some important facts about gender.

- Cross-gender behaviour is universal. In countless species, biologically male animals will adopt behaviour, courtship rituals and mating strategies that parallel those seen in female animals (Roughgarden, 2004). In humans, most children engage in some form of play that violates gender roles.
- Some people who desire to change their gender identity pursue hormonal treatments to change secondary sexual characteristics and sex-reassignment surgery, in which the existing genitalia are altered to be more similar to those of the opposite sex. Surveys of hundreds of people one year after they have undergone such surgery indicate that more than 90 percent of people are satisfied and do not regret the surgery (Green & Fleming, 1990). Sex-reassignment surgery is related to improvements in partner relationships (De Cuypere et al., 2005) and sexual satisfaction (Lief & Hubschman, 1993).

## QUESTIONS

1. Research and name animals in which biologically male animals adopt the roles seen in female animals.
2. If you could adjust the definition of gender dysphoria in the DSM, what else would you include?

What are the norms in our culture today? To answer this question, it is important to gather samples that are representative of the population in terms of age, gender, ethnicity, socioeconomic status and other key characteristics. We will discuss various representative surveys involving thousands of participants throughout this chapter. See table 9.1 for data from one of these large representative surveys. Sometimes participants in these studies are asked to respond to written questions, as people may feel more comfortable describing their behaviour in writing than in discussion with an interviewer. In one recent large-scale representative study (Herbenick et al., 2010b), researchers gathered data over the internet, as it seemed that participants might feel most comfortable with this format. Even with sensitivity to participant comfort, it remains difficult to gather data on how common certain sexual behaviours are. We will discuss this issue in more detail when we discuss paraphilic disorders later in this chapter.

**TABLE 9.1** Participation in selected sexual behaviours in the past year by gender

Behaviour	Male (%)	Female (%)
Masturbation	72	42
Looked at pornography	63	20
Used a sex toy (vibrator or dildo)	15	21
Used internet/phone app to find a partner	7	4
Had an orgasm	92	66

*Source:* Richters et al. (2014).

## Gender and sexuality

Few topics raise as much political debate or personal turmoil as gender differences in sexuality. Across a wide range of indices, men report more engagement in sexual thought and behaviour than do women. Of course, these are averages, there are going to be exceptions and most of these differences are small (Andersen, Cyranowski, & Aarestad, 2000; de Visser et al., 2014). But compared to women, men report thinking about sex, masturbating and desiring sex more often, as well as desiring more sexual partners, having more sexual partners, oral sex, anal sex, having more extramarital affairs, using condoms and engaging in cybersex (Baumeister, Catanese, & Vohs, 2001; Herbenick et al., 2010a; Petersen & Hyde, 2010).

Beyond these differences in sex drive, Petersen and Hyde (2010) have described several other ways in which the genders tend to differ in sexuality. Women tend to report more fear, anxiety and guilt about sex (Petersen & Hyde, 2010) and to be more ashamed of any flaws in their appearance than do men and this shame can interfere with sexual satisfaction (Sanchez & Kiefer, 2007). For women, sexuality appears more closely tied to relationship status than for men (Baumeister, 2000). For example, women tend to report less sexual drive and masturbation when they are not in a relationship; men don't experience the same shift when a relationship ends. Some argue that the DSM pays too little attention to the relational components of human sexuality in describing sexual dysfunction, especially for women. Some propose that there should be a more women-centred definition that includes 'discontent or dissatisfaction with any emotional, physical or relational aspect of sexual experience' (Tiefer, Hall, & Tavis, 2002, pp. 228–229). Among women with symptoms of sexual dysfunction, more than half believe their symptoms are caused by relationship problems (Nicholls, 2008). Men are more likely to think about their sexuality in terms of power than women (Andersen, Cyranowski, & Espindle, 1999).

It is important to acknowledge commonalities as well as differences across genders. In one parallel to men's sexuality, in a survey of more than 1000 women, many reported that their primary motivation for having sex was sexual attraction and physical gratification (Meston & Buss, 2009). It would be an exaggeration to claim that the sole reason women are having sex is to promote relationship closeness.

Nonetheless, given that some gender differences are apparent, debate continues about the reasons for these gender differences. Are they based on cultural prohibitions regarding women's sexuality? Are they based on biological differences? Are they tied to women's greater investment in parenting? It is hard to design research to tease apart cultural and biological influences on sexuality. Intriguingly, though, at least some research suggests that some gender differences are remarkably consistent across cultures. In one study of more than 16 000 people (albeit mostly university students), men in 52 different countries reported that they wanted more partners over the course of a lifetime than did women (Schmitt et al., 2003; Petersen & Hyde, 2010). These findings suggest that biology may shape men's desire for many lifetime partners more than culture does. We know less about the basis for other gender differences in sexuality. There must be some reason for these differences, however. As Baumeister (2000) points out, in a perfect world, wouldn't men and women be well matched on their sexual preferences?

Throughout this chapter, we will see that gender shapes sexual disorders in a number of ways. Women are much more likely to report symptoms of sexual dysfunction than are men,

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As portrayed in the movie *Kinsey*, Alfred Kinsey (shown here) shocked people when he began to interview people to understand more about norms in sexual behaviour.



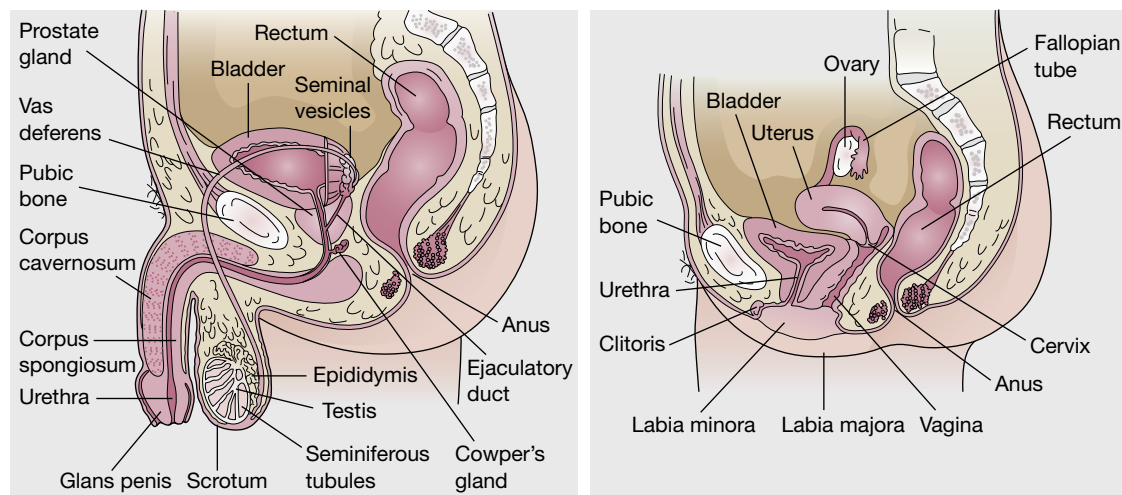
but men are much more likely to meet diagnostic criteria for paraphilic disorder (Dawson, Bannerman, & Lalumière, 2016). Research on basic gender differences in sexuality is needed to understand why there are such major gender differences in sexual diagnoses.

## The sexual response cycle

Many researchers have focused on understanding the **sexual response cycle**. The Kinsey group made breakthroughs in the 1940s by interviewing people about their sexuality (Kinsey, Pomeroy, & Martin, 1948). Masters and Johnson created another revolution in research on human sexuality 50 years ago when they began to gather direct observations and physiological measurements of people masturbating and having sexual intercourse. Most contemporary conceptualisations of the sexual response cycle draw from proposals by Masters and Johnson (1966), which were developed further by Kaplan (1974).

1. **Desire phase.** This phase, introduced by Kaplan (1974), refers to sexual interest or desire, often associated with sexually arousing fantasies or thoughts.
2. **Excitement phase.** During this phase, men and women experience increased blood flow to the genitalia (see figure 9.2 for the sexual anatomy of men and women). In men, this flow of blood into tissues produces an erection of the penis. In women, blood flow creates enlargement of the breasts and changes in the vagina, such as increased lubrication.
3. **Orgasm phase.** In this phase, sexual pleasure peaks in ways that have fascinated poets and the rest of us ordinary people for thousands of years. In men, ejaculation feels inevitable and indeed almost always occurs (in rare instances, men have an orgasm without ejaculating and vice versa). In women, the outer walls of the vagina contract. In both sexes, there is general muscle tension.
4. **Resolution phase.** This last phase refers to the relaxation and sense of wellbeing that usually follow an orgasm. In men there is an associated refractory period during which further erection is not possible. The duration of the refractory period varies across men and even in the same man across occasions. Women are often able to respond again with sexual excitement almost immediately, a capability that permits multiple orgasms.

**FIGURE 9.2** The male and female sexual anatomy

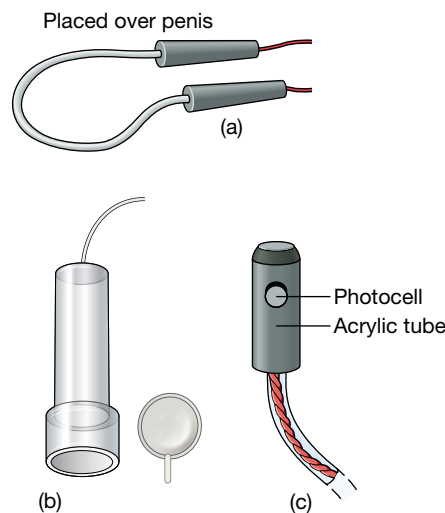


Newer data calls into question the validity of distinguishing the desire and excitement phases for women. Although the model above suggests that desire would precede excitement, many women report that their desire and excitement co-occur and are not distinct (Graham, 2010) and about a third of women report that their desire follows (rather than precedes) physiological arousal (Carvalheira, Brotto, & Leal, 2010).

There is also some question about the way in which Kaplan defined the excitement phase by relying on biological changes. Subjective excitement may not mirror biological excitement for women. Some of the research on this topic has used a device called a **vaginal plethysmograph** to measure women's physiological arousal (see figure 9.3). When blood flow is measured by the vaginal plethysmograph, most women experience a rapid, automatic response to erotic stimuli. But the amount of blood flow to the vagina has little correlation with women's subjective level of desire or excitement (Basson, Brotto, Laan, Redmond, & Utian, 2005). Indeed, many women report little or no subjective excitement when those biological changes happen (Chivers, Seto, Lalumiere, Laan, & Grimbos, 2010). Biological and subjective excitement needs to be considered separately for women, even though they tend to be highly correlated for men.

**FIGURE 9.3**

Behavioural researchers use genital devices for measuring biological sexual arousal. These devices are sensitive indicators of blood flow into the genitalia, a key physiological process in sexual arousal. For men, the penile plethysmograph measures changes in the size of the penis. (a) In one version of the penile plethysmograph, a very thin rubber tube is used. As the penis enlarges with blood, the tube stretches, changing its electrical resistance. (b) Less commonly, a rubber sheath is inserted over the penis and then a chamber is placed over the penis. As the penis enlarges, the volume of air displaced is measured, providing a more accurate measure of change. (c) For women, biological sexual arousal can be measured by a vaginal plethysmograph. This tampon-shaped apparatus can be inserted into the vagina to measure increases in blood flow. Biological arousal may not be associated with subjective arousal or desire for women.



## 9.2 Sexual dysfunctions

**LEARNING OUTCOME 9.2** Explain the symptoms, causes and treatments for sexual dysfunctions.

At its best, sexuality provides a forum for closeness, connection and shared pleasure. Essentially, our sexuality shapes part of our self-concept. Do we please the people we love or, more simply, are we able to enjoy fulfilment from a pleasurable sexual experience? When sexual problems emerge, they can wreak havoc on our self-esteem and relationships (Karney & Bradbury, 1995). Partnerships are likely to suffer if sexual dysfunctions become so severe that the intense satisfaction and tenderness of sexual activity are diminished or, even worse, lost.

We turn now to sexual problems that interfere with sexual enjoyment for many people at some time during their life. We begin by describing the different types of sexual dysfunctions described in the DSM-5. Then we discuss aetiology and treatments for these problems.

## Clinical descriptions of sexual dysfunctions

The DSM-5 divides **sexual dysfunctions** into three categories: those involving sexual desire, arousal and interest; orgasmic disorders; and a disorder involving sexual pain (see table 9.2). Separate diagnoses are provided for men and women. The diagnostic criteria for all sexual dysfunctions specify that dysfunction should be persistent and recurrent and should cause clinically significant distress or problems with functioning. A diagnosis of sexual dysfunction is not made if the problem is believed to be due entirely to a medical illness (such as advanced diabetes or cancer, which can cause erectile problems in men) or to another psychological disorder (such as clinical depression).

**TABLE 9.2** Diagnoses of sexual dysfunction in the DSM-5

Category of dysfunction	Diagnoses in women	Diagnoses in men
Sexual interest, desire and arousal	Female sexual interest/arousal disorder	Male hypoactive sexual desire disorder
Orgasmic disorder	Female orgasmic disorder	Erectile disorder
Sexual pain	Genito-pelvic pain/penetration disorder	Premature ejaculation Delayed ejaculation

One might not expect people to report problems as personal as sexual dysfunction in community surveys. But many people do report these symptoms — the prevalence of occasional symptoms of sexual dysfunction is surprisingly high. Table 9.3 presents data from a survey of more than 20 000 men and women who were asked whether they had experienced various symptoms of sexual dysfunction for at least 2 of the past 12 months (Laumann et al., 2005). More women (43 percent) than men (31 percent) reported symptoms of sexual dysfunction (Laumann, Paik, & Rosen, 1999). Other studies have reported a very similar percentage (about 44 percent) of women who reported symptoms of sexual dysfunction (cf. Bancroft, Loftus, & Long, 2003; Shifren, Monz, Russo et al., 2008). Although participants acknowledged symptoms, clinical diagnoses are not made unless the symptoms cause distress or impairment. When asked whether they are distressed by these symptoms, only 11 to 23 percent of women report experiencing both sexual symptoms and distress over the symptoms.

Beyond a lack of attention to distress or impairment, these surveys typically do not assess how long symptoms have lasted. DSM-5 criteria for sexual dysfunction disorders specify that symptoms must last at least six months. It is pretty common for people to have sexual symptoms for a month. For more than three-quarters of people, these symptoms will remit naturally over time (Mercer et al., 2003).

Although the diagnostic system for sexual dysfunction reflects the stages in the sexual cycle, the problems often don't break out so cleanly in real-life. Oftentimes, people with problems in one phase of a sexual cycle will report problems in another phase. Some of this may just be a vicious circle. For example, men who develop premature ejaculation may begin to worry about sex and then experience problems with sexual desire or sexual arousal (Rowland, Cooper, & Slob, 1996). Beyond the consequences for the individual, sexual problems in one person may lead to sexual problems in the partner. Be aware of this potential for co-occurrence of diagnoses as we review the specific sexual dysfunction disorders defined in the DSM-5.

The pioneering work of the sex therapists William H Masters and Virginia Johnson helped launch a candid and scientific appraisal of human sexuality and for that, we thank them!



TABLE 9.3

Self-reported rates of experiencing symptoms of sexual dysfunction for 2 of the past 12 months by region among 20 000 sexually active adults aged 40 to 80

	Lacked interest in sex	Inability to reach orgasm	Orgasm reached too quickly	Pain during sex	Sex not pleasurable	Trouble lubricating	Trouble maintaining or achieving an erection
<b>Women</b>							
Northern Europe	25.6	17.7	7.7	9.0	17.1	18.4	NA
Southern Europe	29.6	24.2	11.5	11.9	22.1	16.1	NA
Non-European West	32.9	25.2	10.5	14.0	21.5	27.1	NA
Central/South America	28.1	22.4	18.3	16.6	19.5	22.5	NA
Middle East	43.4	23.0	10.0	21.0	31.0	23.0	NA
East Asia	34.8	32.3	17.6	31.6	29.7	37.9	NA
Southeast Asia	43.3	41.2	26.3	29.2	35.9	34.2	NA
<b>Men</b>							
Northern Europe	12.5	9.1	20.7	2.9	7.7	NA	13.3
Southern Europe	13.0	12.2	21.5	4.4	9.1	NA	12.9
Non-European West	17.6	14.5	27.4	3.6	12.1	NA	20.6
Central/South America	12.6	13.6	28.3	4.7	9.0	NA	13.7
Middle East	21.6	13.2	12.4	10.2	14.3	NA	14.1
East Asia	19.6	17.2	29.1	5.8	12.2	NA	27.1
Southeast Asia	28.0	21.1	30.5	12.0	17.4	NA	28.1

**Note:** Non-European West includes Australia, Canada, New Zealand, South Africa and the United States.

**Source:** Laumann et al. (2005).

#### DSM-5

##### DSM-5 criteria for male hypoactive sexual desire disorder

- A. People with male hypoactive sexual desire disorder experience persistently or recurrently deficient (or absent) sexual/erotic thoughts or fantasies and desire for sexual activity. The judgement of deficiency is made by the clinician, taking into account factors that affect sexual functioning, such as age and general and sociocultural contexts of the individual's life.
- B. The symptoms in Criterion A have persisted for a minimum duration of approximately six months.
- C. The symptoms in Criterion A cause clinically significant distress in the individual.
- D. The sexual dysfunction is not better explained by a non-sexual mental disorder or as a consequence of severe relationship distress or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

#### DSM-5

##### DSM-5 criteria for erectile disorder

An individual has erectile disorder if between 75–100 percent of sexual occasions (one of the three criteria must be present for six months):

1. they have an inability to attain an erection
2. they have an inability to maintain an erection for completion of sexual activity
3. they have marked decrease in erectile rigidity.

## DSM-5

### DSM-5 criteria for female sexual interest/arousal disorder

- A. People with female sexual interest/arousal disorder have a lack of, or significantly reduced, sexual interest/arousal, as manifested by at least three of the following:
  - 1. absent/reduced interest in sexual activity
  - 2. absent/reduced sexual/erotic thoughts or fantasies
  - 3. no/reduced initiation of sexual activity, and typically unreceptive to a partner's attempts to initiate
  - 4. absent/reduced sexual excitement/pleasure during sexual activity in almost all or all (approximately 75–100 percent) sexual encounters in identified situational contexts or, if generalised, in all contexts
  - 5. absent/reduced sexual interest/arousal in response to any internal or external sexual/erotic cues (e.g., written, verbal, visual)
  - 6. absent/reduced genital or non-genital sensations during sexual activity in almost all or all (approximately 75–100 percent) sexual encounters (in identified situational contexts or, if generalised, in all contexts).
- B. The symptoms in Criterion A have persisted for a minimum duration of approximately six months.
- C. The symptoms in Criterion A cause clinically significant distress in the individual.
- D. The sexual dysfunction is not better explained by a non-sexual mental disorder or as a consequence of severe relationship distress (e.g., partner violence) or other significant stressors and is not attributable to the effects of a substance/medication or another medical condition.

DSM-5 criteria for sexual dysfunctions include clear guidelines regarding relationship distress. Sexual concerns that arise due to severe relationship distress, such as partner abuse, should not be diagnosed as sexual dysfunctions.

### Disorders involving sexual interest, desire and arousal

The DSM-5 includes three disorders relevant to sexual interest, desire and arousal. **Female sexual interest/arousal disorder** refers to persistent deficits in sexual interest (sexual fantasies or urges), biological arousal or subjective arousal. For men, the DSM-5 diagnoses consider sexual interest and arousal separately. **Male hypoactive sexual desire disorder** refers to deficient or absent sexual fantasies and urges, and **erectile disorder** refers to failure to attain or maintain an erection through the completion of the sexual activity. The clinical case of Gary and Matilda illustrates erectile disorder and the clinical case of Pasquale provides an illustration of hypoactive sexual desire disorder.

## CLINICAL CASE

### Gary and Matilda

When Gary and Matilda sought treatment at a sex therapy clinic, they were a young couple who had been living together for six months and were engaged to be married. Matilda reported that Gary 'hasn't been able to keep his erection after he enters me' for the past two months. Gary would experience initial sexual arousal but then lose his erection almost immediately after entering Matilda. Although they enjoyed sexual intercourse during the beginning of their relationship, the erectile problems started after they moved in together. During the interview, the psychiatrist learned that they had considerable conflict over the amount of time they spent together and over commitment, and at times Matilda had been violent towards Gary. No medical problems appeared to be involved, nor was depression a concern for either Gary or Matilda.

(Adapted from Spitzer, Gibbon, Skodol, Williams, & First, 1994)

### QUESTIONS

- 1. As their therapist, what else would you want to know about this couple?
- 2. What information could you draw together to support a diagnosis?

Among people seeking treatment for sexual dysfunctions, more than half complain of low desire. As table 9.3 shows, women are more likely than men to report at least occasional concerns about their level

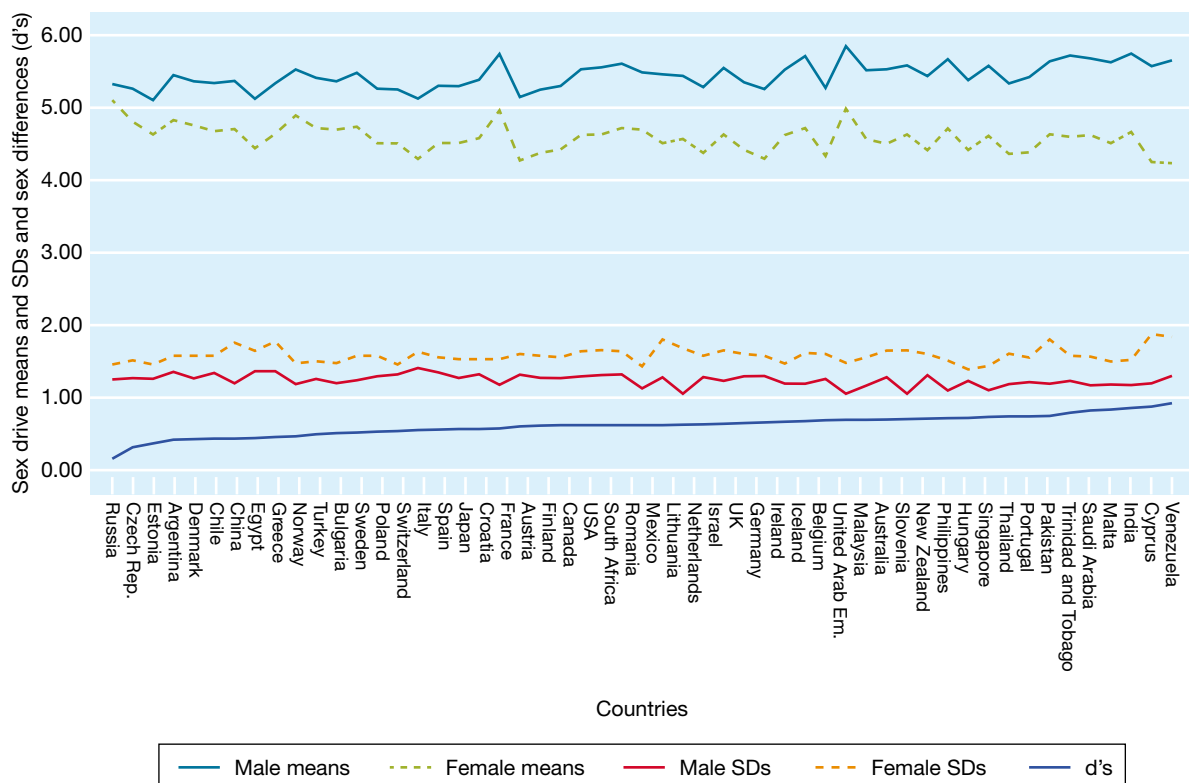
of sexual desire. Postmenopausal women are two to four times as likely as women in their 20s are to report low sexual desire. On the other hand, older women are less likely to be distressed over low sexual desire (Derogatis & Burnett, 2008).

DSM-5 criteria for sexual interest/arousal disorder in women include biologically or subjectively low arousal or desire. Women tend to be more concerned by a lack of subjective desire than by a lack of biological arousal (Basson et al., 2004). Most commonly, women with this disorder report that previously exciting stimuli, such as their partner's touch or a sensual dance, no longer trigger desire (Brotto & Luria, 2014). When laboratory studies are conducted using a vaginal plethysmograph, women who experience a subjective lack of desire often have normative levels of biological response to erotic stimuli (Graham, 2010). This again highlights the distinction between physiological and subjective measures of arousal in women.

Occasional symptoms of erectile disorder are the most common sexual concern among men, with rates ranging from 13 to 28 percent, depending on the country (Laumann et al., 2005). The prevalence of erectile disorder increases greatly with age, with as many as 50 percent of men aged 60 and older reporting at least occasional erectile dysfunction (Rosen, Miner, & Wincze, 2014).

Of all the DSM-5 diagnoses, the sexual interest, arousal and desire disorders, often colloquially referred to as low sex drive, seem the most subjective. How often should a person want sex? And with what intensity? Often, one partner will encourage the other partner to see a clinician if there are irregularities in their intimate and sexual relationship. The hypoactive desire category may owe its existence to the high expectations some people have about being sexual. Data attest to the significance of subjective and cultural factors in defining low sex drive; for example, hypoactive sexual desire disorder is reported more often by American men than by British men despite similar levels of sexual activity across these cultures (Lippa, 2009; see figure 9.4). Cultural norms seem to influence perceptions of how much sex a person 'should' want.

**FIGURE 9.4** Sex drive parameters across 53 nations presented in order of their sex differences in sex drive



Source: Lippa (2009).

## CLINICAL CASE

### Pasquale

Pasquale, a very bright 25-year-old postgraduate student in physics at a leading university, sought treatment for what he called 'sexual diffidence' towards his fiancée. He said he loved his fiancée very much and felt compatible with her in every conceivable way except in bed. There, try as he might and with apparent understanding from his fiancée, he found himself uninterested in initiating or responding to sex. He and his fiancée had attributed these problems to the academic pressures he had faced for the past two years, but a discussion with the therapist revealed that Pasquale had had little interest in sex — either with men or with women — for as far back as he could remember, even when work pressures were not present. He asserted that he found his fiancée very attractive and appealing, but as with other women he had known, he did not feel passion for her.

He had masturbated very rarely in adolescence and did not begin dating until late in university, though he had had many female acquaintances. His general approach to life, including sex, was analytical and intellectual and he described his problems in a very dispassionate and detached way to the therapist. He freely admitted that he would not have contacted a therapist at all were it not for the quietly stated wishes of his fiancée, who worried that his lack of interest in sex would interfere with their future relationship.

After a few individual sessions, the therapist asked the young man to invite his fiancée to a therapy session, which the client readily agreed to do. During a joint session, the couple appeared to be very much in love and looking forward to a life together, despite the woman's concern about Pasquale's lack of sexual interest.

### QUESTIONS

1. If Pasquale had chosen not to see a therapist, how could the situation have impacted on his future relationship with his fiancée?
2. Now that you are aware of Pasquale's potential diagnosis, what are some suggestions that you could make to the couple to help guide them through this situation?

## Orgasmic disorders

The DSM-5 includes separate diagnoses for problems in achieving orgasm for women and men. **Female orgasmic disorder** refers to the persistent absence or reduced intensity of orgasm after sexual excitement. Women differ in their thresholds for orgasm. Although some women have orgasms quickly and without much clitoral stimulation, others need prolonged clitoral stimulation. Therefore, it is not surprising that about one-third of women report that they do not consistently experience orgasms with their partners (Laumann et al., 2005). Female orgasmic disorder is not diagnosed unless the absence of orgasms is persistent and troubling. For many women, enjoying a sense of emotional closeness to their partner is more important than achieving an orgasm (Clark, Thompson, Graham, & Cooper, 2014). About two-thirds of women report that they have faked an orgasm and most say that they did so to try to protect their partner's feelings (Muehlenhard & Shippee, 2010). Many men are unaware (or at least don't report) that their partners don't achieve orgasms (Herbenick et al., 2010a).

## DSM-5

### DSM-5 criteria for female orgasmic disorder

Females can be diagnosed with orgasmic disorder if between 75–100 percent of sexual occasions:

- they experience delay, infrequency or absence of orgasm, or
- they experience markedly reduced intensity of orgasmic sensation.

## CLINICAL CASE

### Anne

Anne was a 26-year-old, attractive woman who sought treatment after beginning her first committed sexual relationship. She reported that she and her partner, Colin, enjoyed their sexual life together. Nonetheless, Colin had begun to express concerns because she had been unable to achieve an orgasm. She sought therapy independently to talk about this concern. In therapy, she described that she had not really enjoyed sexual activities with her former partners and that she had rarely masturbated. Anne disclosed to the therapist that her brother had raped her when she was 12 years old, an event that she had not been able to disclose to anyone in the past. She described harbouring fear about 'letting herself go' after that incident. The therapist provided directed masturbation exercises (described as we review sex therapy interventions below) for Anne to begin to explore and enjoy her body independently, and in sessions they discussed her abuse history and her feelings about sexuality. With the use of a vibrator, Anne was able to begin to enjoy orgasms. As her comfort with her body and sexuality increased, Anne also learned to discuss her sexual preferences more openly with Colin.

(Adapted from Graham, 2014)

### QUESTIONS

1. Are you able to locate or create a 'directed masturbation exercise' sheet to be able to provide to future clients?
2. How would you direct Anne through practising having a conversation about sexual preferences more openly with Colin?

The problem of reaching orgasm is distinct from problems with sexual arousal. As with the clinical case of Anne, many women with orgasmic disorder achieve sexual arousal and enjoy sexual contact, even though they have difficulty reaching orgasm. Indeed, laboratory research has shown that arousal levels while viewing erotic stimuli do not distinguish women with orgasmic disorder from those without orgasmic disorder (Meston & Gorzalka, 1995).

The DSM-5 includes two orgasmic disorders for men: **premature ejaculation**, defined by ejaculation that occurs too quickly, and **delayed ejaculation disorder**, defined by persistent difficulty in ejaculating. The DSM-5 defines 'premature' as less than one minute after the penis is inserted (APA, 2013a, p. 444). One minute was chosen based on cross-national studies showing that the median time to ejaculation is five minutes after penis insertion (Waldinger et al., 2005). As shown in table 9.3, brief periods of symptoms are fairly common, but less than 3 percent of men report symptoms of premature ejaculation lasting six months or more (Segraves, 2010). Delayed ejaculation is the least common sexual dysfunction among men, reported by less than 1 percent of men (APA, 2013a).

## DSM-5

### DSM-5 criteria for premature ejaculation

Premature ejaculation is diagnosed if in between 75–100 percent of sexual occasions there is a tendency to ejaculate during partnered sexual activity within one minute of penile insertion.

## DSM-5

### DSM-5 criteria for delayed ejaculation

Delayed ejaculation is diagnosed if in between 75–100 percent of sexual occasions there is a marked delay, infrequency or absence of orgasm.

## Sexual pain disorder

The major symptom of **genito-pelvic pain/penetration disorder** is persistent or recurrent pain during intercourse. Although some men experience recurrent pain during sex, very few men seek treatment for it. For this reason, the DSM-5 criteria focus only on women. Sexual pain is a very common concern heard by gynaecologists (Leiblum, 1997). Women with this disorder often experience vaginismus, defined by involuntary muscle spasms of the outer third of the vagina to a degree that makes intercourse impossible (Binik, 2010). A first step in diagnosing genito-pelvic/penetration disorder is ensuring that the pain is not caused by a medical problem, such as an infection or by a lack of vaginal lubrication due to low desire or postmenopausal changes.

### DSM-5

#### DSM-5 criteria for genito-pelvic pain/penetration disorder

People with genito-pelvic pain/penetration disorder experience persistent or recurrent difficulties with at least one of the following.

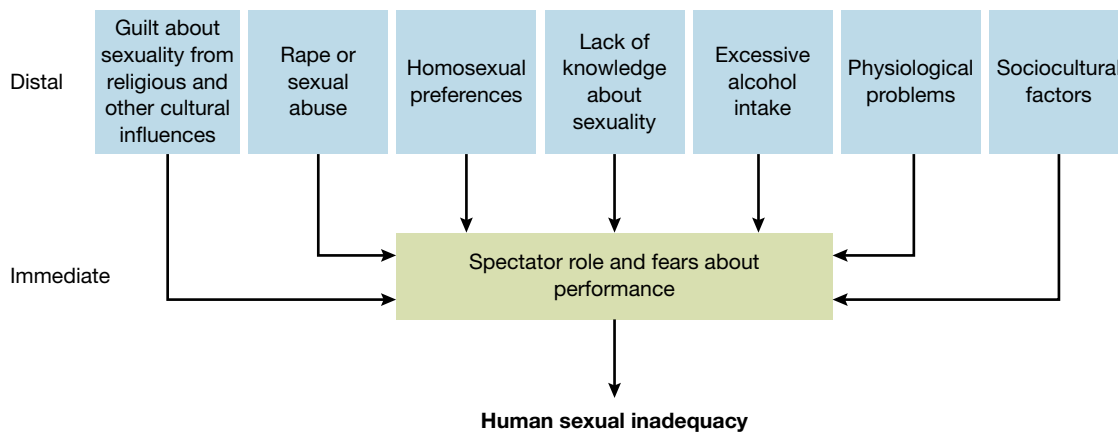
1. Inability to have vaginal/penetration during intercourse
2. Marked vulvar, vaginal or pelvic pain during vaginal penetration or intercourse attempts
3. Marked fear or anxiety about pain or penetration
4. Marked tensing of the pelvic floor muscles during attempted vaginal penetration

Most women diagnosed with this disorder experience sexual arousal and can have orgasms from manual or oral stimulation that does not involve penetration. Women who experience pain when attempting sexual intercourse show normative sexual arousal to films of oral sex, but, not surprisingly, their arousal declines when they watch a depiction of intercourse (Wouda et al., 1998), which suggests that there is a psychological connection to consider. We will further discuss this psychological component shortly.

## Aetiology of sexual dysfunctions

In their widely acclaimed book *Human Sexual Inadequacy*, Masters and Johnson (1970) drew on their case studies to publish a theory of why sexual dysfunctions develop. Masters and Johnson differentiated immediate and distal causes of sexual dysfunction (see figure 9.5). In their model, the two immediate causes are fears about performance and the adoption of a **spectator role**. Fears about performance involve concerns with how one is 'performing' during sex. Spectator role refers to being an observer rather than a participant in a sexual experience. These two related concerns could both impede natural sexual responses. These immediate causes of sexual dysfunctions were hypothesised to have one or more historical antecedents, such as sociocultural influences, biological causes or sexual abuse. Masters and Johnson emphasised that sexual functioning is complex and multifactorial and set the stage for researchers to study these risk factors. We now turn to research on the causes of sexual dysfunctions. Figure 9.6 summarises factors related to sexual dysfunctions.

**FIGURE 9.5** Distal and immediate causes of human sexual inadequacies, according to Masters and Johnson



**FIGURE 9.6** Predictors of sexual functioning

	Successful sexual functioning	Poor sexual functioning
Psychological factors	Good emotional health Attraction towards partner Positive attitude towards partner Positive sexual attitude	Depression or anxiety disorders Focus on performance Too much routine Poor self-esteem Uncomfortable environment for sex Rigid, narrow attitude towards sex Negative thoughts about sex
Physical factors	Good physical health Regular appropriate exercise Good nutrition	Smoking Heavy drinking Cardiovascular problems Diabetes Neurological diseases Low physiological arousal SSRI medications Antihypertensive medication Other drugs
Social and sexual history factors	Positive sexual experiences in past Good relationship with partner Sexual knowledge and skills	Rape or sexual abuse Relationship problems, such as anger or poor communication Long periods of abstinence History of hurried sex

*Source:* Adapted from Wincze and Barlow (1997).

## Biological factors

As noted earlier, a first step in making a diagnosis of sexual dysfunction is to rule out medical diseases as the cause. The DSM-5 includes separate diagnoses for sexual dysfunctions that are caused by medical illnesses. Some have criticised this division in the diagnoses because sexual dysfunctions often have some biological and some psychological contributions. Biological causes of sexual dysfunctions can include diseases such as diabetes, multiple sclerosis and spinal cord injury; heavy alcohol use before sex; chronic alcohol dependence; and heavy cigarette smoking (Bach, Wincze, & Barlow, 2001; Nagaraj, Pai, Rao,

Rao, & Goyal, 2009). Laboratory tests of hormone levels are a routine part of assessment of sexual dysfunctions for men and postmenopausal women (Bartlik & Goldberg, 2000; Buvat et al., 2010) because sexual dysfunctions can be caused by either low levels of testosterone or oestrogen or by the high levels induced by chronic use of anabolic steroids or testosterone supplements. Certain medications, such as antihypertensive drugs and especially selective serotonin reuptake inhibitor (SSRI) antidepressant drugs (e.g., Prozac and Zoloft), have effects on sexual function, including delayed orgasm, decreased libido and diminished lubrication (Segraves, 2003). Erectile symptoms are often related to an incipient vascular disorder (Wylie & MacInnes, 2005). Beyond these medical causes, some biological factors may be specific to certain sexual dysfunctions. As one example, some women who experience genito-pelvic pain/penetration disorder appear to have a neurologically based super-sensitivity to pain (van Lankveld et al., 2010).

### **Psychosocial factors**

Some sexual dysfunctions can be traced to rape or sexual abuse. Childhood sexual abuse is associated with diminished arousal and desire and, among men, with double the rate of premature ejaculation (Laumann, Paik, & Rosen, 1999). See focus on discovery 9.3, later in this chapter, for a discussion of childhood sexual abuse and its repercussions. Beyond the role of traumatic experiences, it is important to consider the benefits of positive experiences — many people with sexual problems lack knowledge and skill because they have not had opportunities to learn about their sexuality.

The more a couple experiences relationship issues, the more likely they will experience problems with sexual arousal and pleasure (Burri & Spector, 2011). For women, concerns about a partner's affection appear particularly correlated with sexual satisfaction (Nobre & Pinto-Gouveia, 2008). For people who tend to be anxious about their relationships, sexual problems may exacerbate underlying worries about relationship security (Birnbaum, Reis, Mikulincer, Gillath, & Orpaz, 2006). As one might expect, people who are angry with their partners are less likely to want sex (Beck & Bozman, 1995). Even in couples who are satisfied with other realms of the relationship, poor communication can contribute to sexual dysfunction. For any number of reasons, including embarrassment, worry about the partner's feelings or fear, one lover may not tell the other about preferences even if a partner is engaging in unstimulating or even aversive behaviours during sex.

Depression and anxiety increase the risk of sexual dysfunctions. People who are depressed are more than twice as likely as non-depressed people to have a sexual dysfunction (Angst, 1998). People with panic disorder, who are often fearful of physical sensations like rapid heart rate and sweating, are also at risk for sexual dysfunction (Sbrocco, Weisberg, Barlow, & Carter, 1997). Anxiety and depression are particularly comorbid with sexual pain (Meana, Binik, Khalife, & Cohen, 1998) and with female sexual interest/arousal disorder (Forbes & Schniering, 2013).

Beyond the detrimental effects of depression and anxiety, several studies suggest that low general physiological arousal can interfere with specific sexual arousal. Meston and Gorzalka (1995) examined the role of arousal by assigning women to exercise or no-exercise conditions and then asking women to watch erotic films. Consistent with the positive role of higher arousal, exercise facilitated sexual arousal. No wonder, then, that exhausted couples, turning to sex after a full day of work, parenting, socialising and other roles, can encounter problems with sexuality. Too much stress and exhaustion clearly impede sexual functioning (Morokoff & Gilliland, 1993).

Negative cognitions, such as worries about pregnancy or STIs, negative attitudes about sex or concerns about the partner, can interfere with sexual functioning (Reissing, Binik, & Khalife, 1999). Intrusive negative thoughts about their weight or appearance impinge on the enjoyment of sex for many women (Pujols, Seal, & Meston, 2010). But as Masters and Johnson first suggested, cognitions concerning sexual performance are particularly important for both men and women (Carvalho & Nobre, 2010). Consider the idea that variability in sexual performance is common; a stressful day, a distracting context, a relationship concern or any number of other issues may diminish sexual responsiveness. The key issue may be how people think about their diminished physical response when it happens. One theory is that people who blame themselves for decreased sexual performance will be more likely to develop recurrent sexual problems.

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Depression and anxiety increase the risk of sexual dysfunctions.



In a test of the role of self-blame and erectile dysfunction, researchers asked 52 male participants to watch erotic videos (Weisberg, Brown, Wincze, & Barlow, 2001). During the videos, their sexual arousal (penile circumference) was measured using a **penile plethysmograph** (see figure 9.3). Regardless of their actual arousal, the men were given false feedback that the size of their erection was smaller than that typically measured among aroused men. Men were randomly assigned to receive two different explanations for this false feedback. In the first, they were told that the films did not seem to be working for most men (external explanation). In the second, they were told that the pattern of their responses on questionnaires about sexuality might help explain the low arousal (internal explanation). After receiving this feedback, the men were asked to watch one more film. The men who were given an internal explanation reported less arousal and also showed less physiological arousal during this film than those given an external explanation. These results, then, support the idea that people who blame themselves when their body doesn't perform will experience diminished subsequent arousal. Needless to say, men in this study were carefully debriefed after the experiment!

In considering the source of negative cognitions, Masters and Johnson found that many of their sexually dysfunctional patients had learned negative views of sexuality from their social and cultural surroundings. For example, some religions and cultures may discourage sexuality for the sake of pleasure, particularly outside marriage. Other cultures may disapprove of sexual initiative or behaviour among women, other than for the sake of procreation. One female patient suffering from a lack of sexual desire, for example, had been taught as she was growing up not to look at herself naked in the mirror and that intercourse was reserved for marriage and then only to be endured for purposes of having children. Guilt about engaging in sexual behaviour varies by cultural group and can inhibit sexual desire (Woo, Brotto, & Gorzalka, 2011).

## Treatments of sexual dysfunctions

Given the complex matrix of factors that promote healthy sexual functioning, it is perhaps no surprise that sex therapists often draw on a rich array of strategies to help their clients. Sexual dysfunctions

are often embedded in a distressed relationship, so that many therapists work from a systems perspective in which a sexual problem is viewed as one expression of relationship problems (Wylie, 1997). Troubled couples usually need training in non-sexual communication skills. Some therapists focus on non-sexual issues, such as difficulties with in-laws or with child rearing — either in addition to or instead of interventions directly focused on sex. For some couples, planning romantic events together, such as a date night, is recommended as a way to restore closeness and intimacy (Kaplan, 1997). For women with sexual dysfunctions occurring in the context of relationship distress, behavioural couples therapy has been found to improve many aspects of sexual functioning (Zimmer, 1987).

Beyond couples therapy, many techniques have been developed to target specific sexual concerns. The pioneering work of Masters and Johnson (1970) in the treatment of sexual dysfunctions is described in focus on discovery 9.2. Over the past decades, therapists and researchers have elaborated on this work and now many different sex therapy interventions are available, several of which we will describe. A therapist may choose only one technique for a given case, but the multifaceted nature of sexual dysfunctions often requires the use of a combination of techniques. These approaches are generally suitable for treating sexual dysfunctions in homosexual as well as heterosexual clients. We will begin by considering different cognitive and behavioural interventions, which have been supported across 20 randomised controlled trials and appear particularly helpful for women with sexual interest/arousal disorder or orgasmic disorder (Frühauf, Gerger, Schmidt, Munder, & Barth, 2013). After discussing cognitive-behavioural approaches, we will turn to physical and medical treatments.

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Many therapists view a sexual problem as one expression of relationship problems, and so offer couples therapy as a treatment option.



### **Masters and Johnson's therapy for sexual dysfunctions**

In their book *Human Sexual Inadequacy*, Masters and Johnson (1970) reported the successful results of one of the first sex therapy programs, which they had offered to almost 800 clients. Each couple had travelled to St. Louis and spent two weeks participating in intensive therapy during the day and completing sexual homework in a hotel at night.

Couples were always seen by one male and one female therapist. For the first two days, couples completed an assessment of their medical, social and sexual history. Sometimes the couple discussed sex for the first time at the clinic. During the assessment and first few days of treatment, sexual intercourse was forbidden.

On the third day, the therapists began to offer feedback about the sources of problems. If a person had negative attitudes towards sex, this issue would be addressed. But the basic emphasis was on relationship problems rather than the individual difficulties of either partner. Whatever the problem, the couple was encouraged to see it as their mutual responsibility. At the same time, the clients were introduced to the idea of the spectator role. They were told, for example, that a male with erectile problems — and often his partner as well — usually worries about how well he is doing and that this pattern of observing the state of the erection, though totally understandable, blocks his natural responses and interferes with sexual enjoyment.

At the end of the third day, the couple was asked to engage in sensate-focus. The couple was instructed to choose a time when both partners felt a sense of warmth and compatibility. During sensate-focus exercises, the couple was instructed not to have intercourse. Indeed, initially they were instructed not to touch each other's genitalia. Rather, they were instructed to undress and give each other pleasure by touching each other's bodies. The co-therapists appointed one marital partner to do the first pleasuring; the partner who was 'getting' was simply allowed to enjoy being touched. The one being touched was not required to feel a sexual response and was responsible for immediately telling the partner if something became uncomfortable. Then the roles were switched. The sensate-focus assignment usually promoted contact, constituting a first step towards re-establishing sexual intimacy. Most of the time, partners began to realise that their physical encounters could be intimate and pleasurable without necessarily being a prelude to sexual intercourse.

On the next evening, the partner being pleasured was instructed to give specific direction by guiding his or her partner's hand to regulate pressure and rate of stroking. Touching of genitals and breasts was now allowed, but still no intercourse. After two days of sensate-focus, treatment began to be tailored to specific problems. For example, guided masturbation exercises (covered below) might be introduced to address female orgasmic disorder.

Many therapists continue to use the Masters and Johnson techniques, although success rates have tended to be lower than those originally reported (Segraves & Althof, 1998). The lower success rates may be attributed to the couples that are seeking treatment these days rather than to the therapy itself. As sex therapy has become more popular and a broader range of couples have begun to seek treatment, treatment providers see couples today with poorer marital relationships than those who sought treatment in the early years of sex therapy.

#### **QUESTIONS**

1. What do you see to be the benefits of intensive sex therapy?
2. Why do you think the avoidance of sexual intercourse is required for the first few days of intensive sex therapy?

### **Anxiety reduction and psychoeducation**

Many clients with sexual dysfunctions require gradual and systematic exposure to anxiety-provoking aspects of the sexual situation. Systematic desensitisation and in-vivo (real-life) desensitisation have been employed with some success (Wolpe, 1958), especially when combined with skills training. For example, a woman with genito-pelvic pain/penetration disorder might first receive psychoeducation about her body,

be trained in relaxation and then practise inserting her fingers or dilators into her vagina, starting with inserting smaller dilators and working up to larger ones (Leiblum, 1997). Such programs have been shown to be helpful for many women with sexual pain disorder (ter Kuile & Reissing, 2014).

Psychoeducation programs about sexuality also do plenty to reduce anxiety. Therapists often assign written materials and show clients explicit videos demonstrating sexual techniques. Several studies have now shown that psychoeducation can be as effective as systematic desensitisation for male erectile disorder and for women with orgasmic disorder or low sexual arousal (Emmelkamp, 2004; Melnik, Soares, & Nasello, 2008).

For the treatment of premature ejaculation, anxiety-reduction techniques sometimes have a different focus. Anxiety about ejaculating too soon may be a natural result of an overemphasis on intercourse as a sole focus of sexual behaviour. Sex therapists advise couples to expand their repertoire of activities to include other ways of gratifying their partner both before and after the man has climaxed, such as oral or manual manipulation. When the exclusive focus on penile insertion is removed, anxieties about sex often diminish enough to enhance the man's control over ejaculation.

### **Procedures to change attitudes and thoughts**

The sensate-focus exercises described in focus on discovery 9.2 are a way of helping the person focus on physical sensations as a counter to the destructive tendency to think about one's performance or attractiveness during sex. Other cognitive interventions are designed to challenge the self-demanding, perfectionistic thoughts that often cause problems for people with sexual dysfunctions. A therapist might try to reduce the pressure a man with erectile dysfunction feels by challenging his belief that intercourse is the only true form of sexual activity. Women who are hypercritical of their appearance might be coached to consider more positive ways of viewing their bodies and their sexuality.

### **Communication training**

Encouraging partners to communicate their likes and dislikes to each other has been shown to be helpful for a range of sexual dysfunctions (Rosen, Leiblum, & Spector, 1994). Communication training also exposes partners to potentially anxiety-provoking material, such as expressing sexual preferences, which allows for a desensitising effect. Skills and communication training is particularly warranted when sexual dysfunction is specific to a given relationship and was not a concern with previous partners.

### **Directed masturbation**

Directed masturbation was devised by LoPiccolo and Lobitz (1972) to enhance women's comfort with and enjoyment of their sexuality. The first step is for the woman to carefully examine her nude body, including her genitals and to identify various areas with the aid of diagrams. Next, she is instructed to touch her genitals and to find areas that produce pleasure. Then she increases the intensity of masturbation using erotic fantasies. If orgasm is not achieved, she is to use a vibrator in her masturbation. Finally, her partner enters the picture, first watching her masturbate, then doing for her what she has been doing for herself and finally having intercourse in a position that allows him to stimulate the woman's genitals manually or with a vibrator. As illustrated in the clinical case of Anne, directed masturbation has been shown to be helpful in treating orgasmic disorder (O'Donohue, Dopke, & Swingen, 1997), particularly when women have a lifelong inability to experience orgasm, with 60–90 percent of that subgroup achieving orgasm post-treatment (ter Kuile, Both, & van Lankveld, 2012). It is also helpful in the treatment of low sexual desire (Renshaw, 2001).

### **Other physical treatments**

For the treatment of female orgasmic disorder, couples may be taught specific sexual positions that increase the amount of clitoral stimulation. For the treatment of premature ejaculation, the squeeze technique is often used, in which a partner is trained to squeeze the penis in the area where the head and shaft meet to rapidly reduce arousal. This technique is practised without insertion and then during insertion, the penis is withdrawn and the squeeze is repeated as needed. In a similar approach, men are taught

to withdraw their penis as needed during intercourse, so as to reduce arousal. Initial success rates are high (60–90 percent), but some relapse takes place over time (Polonsky, 2000).

## Medications

There has been a huge increase in people taking medications to address sexual concerns. As previously mentioned, there are psychological and interpersonal components of many sexual problems, so providing a blended approach of psychological and pharmacological intervention is currently the best recommended practice as the blended approach appears to produce better outcomes (Frühauf et al., 2013). For example, although premature ejaculation is often treated with medication alone, psychotherapy can help men regain confidence after experiences of these symptoms (Althof, 2014). Despite these caveats, medical treatments for sexual dysfunction, typically offered without psychotherapy, are popular.

### Antidepressant drugs

Antidepressant drugs have been found to be helpful when depression appears to contribute to diminished sex drive. These drugs, particularly SSRIs, have also been found to be helpful in the treatment of premature ejaculation in a series of studies (Althof et al., 2010). A complicating factor, though, is that some antidepressants themselves interfere with sexual responsiveness! Sometimes a second medication is used to counteract the sexual side effects of the first; for example, bupropion (Wellbutrin) has been shown to help address the libido problems caused by SSRIs (Seagraves, 2003).

### PDE-5 inhibitors

The most common intervention for erectile disorder is a phosphodiesterase type 5 (PDE-5) inhibitor, such as sildenafil (Viagra), tadalafil (Cialis) or vardenafil (Levitra). PDE-5 inhibitors relax smooth muscles and thereby allow blood to flow into the penis, creating an erection during sexual stimulation but not in its absence (Eardley et al., 2010). PDE-5 inhibitors are taken one hour before sex and the effects last about four hours. Although some men stop taking these medications due to side effects such as headaches and indigestion, most men will tolerate the side effects to gain relief from their sexual symptoms. Indeed, worldwide sales of PDE-5 medications was valued at \$4.3 billion (USD) in 2012 (PR Newswire, 2013). PDE-5 inhibitors may be dangerous for men with cardiovascular disease and this is a concern since erectile dysfunction is often comorbid with cardiovascular disease.

In a summary of 27 treatment studies, about 83 percent of men who took sildenafil were able to successfully have intercourse compared to about 45 percent of men who were assigned to a placebo condition (Fink, Mac Donald, Rutks, Nelson, & Wilt, 2002). Some men continue to experience intermittent erectile dysfunction on PDE-5 inhibitors, and so again, a blended approach of combining both psychological and medicinal treatment approaches is recommended (Melnik, Soares, & Nasello, 2008). Several trials of PDE-5 inhibitors have been conducted for treatment of sexual dysfunction in women, but the results are not promising (Laan, Everaerd, & Both, 2005).

There has been a huge increase in people taking medications to address sexual concerns.



## 9.3 The paraphilic disorders

**LEARNING OUTCOME 9.3** Explain the symptoms, causes and treatments for paraphilic disorders.

The DSM-5 defines the **paraphilic disorders** as recurrent sexual attraction to unusual objects or sexual activities lasting at least six months. In other words, there is a deviation (*para*) in what the person is attracted to (*philia*). DSM differentiates the paraphilic disorders based on the source of arousal; for example, it provides one diagnostic category for people whose sexual attractions are focused on causing pain and another diagnostic category for people whose attractions are focused on children (see table 9.4). Large surveys of people who volunteer for research on sexuality and health have shown that many people occasionally fantasise about some of the activities we will be describing. Voyeuristic attractions may be particularly common: about 50 percent of men report fantasies of watching unsuspecting naked women (Hanson & Harris, 1997). About 7.7 percent reported that they had been aroused by spying on others having sex, 7 percent reported that they had engaged in sadomasochistic sex at least once and 3.1 percent reported that they had been aroused by exposing their genitalia to a stranger at least once during their lifetime (Långström & Seto, 2006; Vanwesenbeeck, Bakker, & Gesell, 2010). By January 2016, the website fetlife.com, a social network site for people interested in sadomasochistic and fetishistic sex, listed 5 563 551 members. *Fifty Shades of Grey*, a book describing a sadomasochistic relationship, became one of the best-selling books of all time, with over 125 million copies sold worldwide by 2015 (Flood, 2015).

**TABLE 9.4** Paraphilic disorders included in DSM

DSM-5 diagnosis	Object of sexual attraction
Fetishistic disorder	An inanimate object or non-genital body part
Transvestic disorder	Cross-dressing
Paedophilic disorder	Children
Voyeuristic disorder	Watching unsuspecting others undress or have sex
Exhibitionistic disorder	Exposing one's genitals to an unwilling stranger
Frotteuristic disorder	Sexual touching of an unsuspecting person
Sexual sadism disorder	Inflicting pain
Sexual masochism disorder	Receiving pain

As some of these behaviours become more common, considerable debate has emerged about whether it is appropriate to diagnose some of the paraphilias. In 2009, the Swedish National Board of Health and Welfare decided to remove some of the paraphilic diagnoses. Fetishistic disorder, sexual sadism disorder, sexual masochism disorder and transvestic disorder are no longer included in their psychiatric classification system (Långström, 2010). The board reasoned that many people practise variant sexual behaviours safely with consenting adult partners and do not experience any distress or impairment as a result (Richters, de Visser, Rissel, Grulich, & Smith, 2008). The DSM-5 retains the labels, but the word *disorder* is added to the title of these diagnoses to emphasise that the diagnoses are to be considered only when the sexual attractions cause marked distress or impairment or when the person engages in sexual activities with a non-consenting person.

Impairment and engagement of non-consenting others are important boundaries between normative and problematic sexual behaviour. For some sexual behaviours, though, these dimensions rarely apply. For example, transvestic disorder does not typically involve non-consenting others and it rarely leads to impairment; the diagnosis of this disorder typically rests on the presence of distress. In focus on

discovery 9.1, we noted that the diagnostic criteria that rely on distress about sexual desires and behaviours are illogical. The person who cross-dresses for sexual gratification and accepts the behaviour won't meet the diagnostic criteria. In contrast, the person who feels guilty and ashamed because he or she has internalised stigma about this behaviour is diagnosable. Because transvestic behaviour so rarely leads to impairment or involves non-consenting others, we do not discuss transvestic disorder further here.

## CLINICAL CASE

### Kevin

Kevin and Mariko sought marital therapy after Mariko learned that Kevin had a long history of voyeurism. Mariko had been startled to walk into their guest room and find him viewing the neighbour with binoculars while masturbating. Upon confrontation, Kevin shared with his wife that he had felt intense and uncontrollable urges to watch strangers undress since his early adolescence.

Kevin and Mariko reported that they had been married for 20 years and that throughout the duration of their relationship, neither had found their sexual life very satisfying. Mariko was concerned that he rarely initiated sexual contact with her, and indeed, in an individual session, Kevin reported that he preferred watching strangers to having sex with his wife. He had never found sex with a consenting partner to be as exciting as the forbidden.

Kevin had tried different strategies to gain control over his voyeuristic urges, including reading self-help books and attending a support group, with no success. He reported that he came from an extremely strict family and had been teased relentlessly by his father. Although his desire to watch strangers haunted him, he had felt too ashamed to discuss his sexual preferences with anyone in the past. His sense of sexual detachment appeared to be part of a broader pattern of emotional distance and lack of disclosure with others in his life. In therapy, he began to explore the sense of social rejection that he had experienced since early childhood. As his wife learned of his past, they achieved a stronger emotional bond, which freed them to discuss their sexuality more openly. As their sex life improved, Kevin reported that his desire to watch others undress faded. (Adapted from Kleinplatz, 2014)

### QUESTIONS

1. What are some aspects of sexuality that Kevin and Mariko should discuss?
2. Do you think that the therapist could have taken another approach? If so, what?

Accurate prevalence statistics are not available for the paraphilic disorders. Research is limited by the lack of structured diagnostic interviews to reliably assess these conditions (Krueger, 2010b) and even more by the reluctance of many people with paraphilias to reveal their proclivities. Because some people with paraphilic disorders seek non-consenting partners or otherwise violate people's rights in offensive ways (as we will see in exhibitionistic and paedophilic disorders), these disorders can have legal consequences. But statistics on arrests are likely to be underestimates because many crimes go unreported and some paraphilias (e.g., voyeuristic disorder) involve an unsuspecting victim. The data do indicate, however, that most people with paraphilic disorders are male and heterosexual; even with paedophilic disorder and sexual masochism disorder, which occur in noticeable numbers of women, men vastly outnumber women. The onset for many of the paraphilic disorders, including fetishistic disorder, voyeuristic disorder and exhibitionistic disorder, typically occurs during adolescence. The onset of sexual sadism disorder and sexual masochism disorder tends to occur by early adulthood.

Most people with a paraphilic disorder meet the criteria for other paraphilic disorders. In samples of those arrested for their paraphilic behaviours, more than two-thirds meet the criteria for DSM mood disorders and anxiety and substance abuse disorders are also common (Kafka & Hennen, 2002). Here we provide a clinical description of the paraphilic disorders. As we describe the symptoms, we briefly describe the epidemiology of these disorders.

## DSM-5

### DSM-5 criteria for fetishistic disorder

- A person has fetishistic disorder if, for at least six months, they have recurrent and intense sexually arousing fantasies, urges or behaviours involving the use of non-living objects or non-genital body parts.
- These symptoms cause significant distress or impairment in functioning.
- The sexually arousing object(s) are not limited to articles of clothing used in cross-dressing or to devices designed to provide tactile genital stimulation, such as a vibrator.

## Fetishistic disorder

The key feature of **fetishistic disorder** is a reliance on an inanimate object or a non-genital part of the body for sexual arousal. A fetish refers to the object of these sexual urges, such as women's shoes or feet. The person with fetishistic disorder, almost always a man, has recurrent and intense sexual urges towards these fetishes and the presence of the fetish is strongly preferred or even necessary for sexual arousal. Clothing (especially underwear), leather and articles related to feet (stockings, women's shoes) are common fetishes. Beyond non-living objects, some people focus on non-sexual body parts, such as hair, nails, hands or feet, for sexual arousal. Because there is no evidence of a difference in the aetiology or consequences of a boot fetish compared to a foot fetish, the DSM-5 includes a reliance on non-sexual body parts for sexual arousal under the diagnosis of fetishistic disorder.

## CLINICAL CASE

### Cameron

Cameron, a single, 32-year-old male photographer, sought treatment for his concern that he was more attracted to women's shoes than to the women themselves. Cameron reported that he remembered being excited by pictures of women in their shoes at age seven. At age 13 he reached orgasm by masturbating while imagining women in their shoes. He began to steal shoes from his sister to use while masturbating. As he grew older, he would sneak into women's rooms and steal their shoes. He began to have intercourse at age 18 and his preferred partner was a prostitute whom he asked to wear high-heeled shoes while they had sex. He found that he preferred masturbating into stolen shoes more than sexual intercourse. He avoided dating because he feared the scorn that his focus on shoes might provoke. He had begun to experience significant depression over the ways in which his sexual behaviour was limiting his social life. (Adapted from Spitzer et al., 1994)

### QUESTIONS

1. What are the features of the case that suggest Cameron is distressed by his behaviour?
2. What are some ways that Cameron can begin to discuss his fetish with future sexual partners?

The person with fetishistic disorder feels a compulsive attraction to the object; the attraction is experienced as involuntary and irresistible. The exclusive and very special status the object occupies as a sexual stimulant distinguishes fetishistic disorder from the ordinary attraction that, for example, high heels may hold for heterosexual men in Western cultures. The person with a boot fetish must see or touch a boot to become aroused and the arousal is overwhelmingly strong when a boot is present. Some carry on their fetishism by themselves in secret by fondling, kissing, smelling, gazing at the adored object or using the fetish as they masturbate. Others can only reach orgasm if a partner dons the fetish and the resultant distress of the partner is one way that this syndrome reaches a diagnosable level.

## Paedophilic disorder and incest

According to the DSM, **paedophilic disorder** (*pedes* is Greek for ‘child’) is diagnosed when adults derive sexual gratification through sexual contact with pre-pubertal children or when their recurrent and intense desires for sexual contact with pre-pubertal children cause distress either for themselves or others. DSM-5 criteria specify that the offender be at least 16 years old and at least 5 years older than the child. Some may meet diagnostic criteria in the absence of any actions, if their urges are provoking distress. As in most paraphilias, a strong attraction impels the behaviour. Sometimes a man with paedophilic disorder is content to stroke the child’s hair, but he may also manipulate the child’s genitalia, encourage the child to manipulate his and, less often, attempt penile insertion. The molestations may be repeated over a period of weeks, months or years. The National Crime Authority of Australia (NCA) defines ‘paedophiles’ as adults who act on their sexual preference for children, and ‘child’ refers to anyone below the age of consent in each Australian state and territory (Miller, n. d., p. 2).

People with paedophilic disorder generally molest children whom they know, such as neighbours or friends of the family. Most with paedophilic disorder do not engage in violence other than the sexual act, although when they do become violent, it is often a focus of lurid stories in the media. Because overt physical force is seldom used in paedophilic disorder, the child molester often denies that he is actually forcing himself on his victim. Despite molesters’ distorted beliefs, child sexual abuse inherently involves a betrayal of trust and other serious negative consequences (see focus on discovery 9.3 for a discussion of these consequences). Paedophilia is one of the most common paraphilias and is most likely to be seen in treatment because of its harmful and illegal nature (O’Grady, 2001).

What are the demographic characteristics of people who meet the criteria for paedophilic disorder? Up to half are adolescent males (Morenz & Becker, 1995). People with paedophilic disorder can be straight or gay, though most are heterosexual. Most older heterosexual men with paedophilic disorder are or have been married.

### DSM-5

#### DSM-5 criteria for paedophilic disorder

- A person has paedophilic disorder if, for at least six months, they experience recurrent and intense, sexually arousing fantasies, urges or behaviours involving sexual contact with a prepubescent child.
- The person will have acted on these urges, or the urges and fantasies cause marked distress or interpersonal problems.
- Paedophilic disorder is diagnosed if the person is at least 16 years old and at least 5 years older than the child.

Sexual arousal in response to pictures of young children can be measured by the penile plethysmograph and in one meta-analysis of studies involving 28 972 sexual offenders, arousal as measured in this way was one of the strongest predictors of repeated sexual offences (Hanson & Bussiere, 1998). Penile plethysmograph indices may be helpful in predicting future offences when sexual offenders deny an interest in young children (Blanchard, 2010). Nonetheless, arousal in response to pictures of children is not a perfect predictor of paedophilic disorder. Many men who are conventional in their sexual interests and behaviour can be sexually aroused by erotic pictures of children. In a study using both self-report and penile plethysmography, one-quarter of men drawn from a community sample showed or reported arousal when viewing sexually provocative pictures of children (Hall, Hirschman, & Oliver, 1995). Indeed, across studies, 3–9 percent of men describe having experienced at least one sexual fantasy involving a child (Seto, 2009). Consider this quote from a man about recognising that he held sexual attractions to children.

That was the year when Father Smith hit the front page of every newspaper my parents brought home with them. A new evil was born from an inferno of horror and fear and its name was Paedophilia. I had never heard that word before. Father Smith was a paedophile and as the newspaper explained,

a paedophile was a person who had an attraction to children. Father Smith had sex with young boys. The only people I seemed to be attracted to were boys. At first a wave of utter disbelief and confusion passed over me. There is no way on God's green earth I would do anything like the horror Father Smith perpetrated on those boys. (Goode, 2010, pp. 6–7)

### FOCUS ON DISCOVERY 9.3

#### **The effects of paedophilic disorder: outcomes after childhood sexual abuse**

In one major community survey, about 20 percent of women and 5 percent of men reported experiencing some form of childhood sexual abuse (CSA) (Afifi et al., 2008). A child abuser is usually not a stranger. He may be a father, an uncle, a brother, a teacher, a coach, a neighbour or even a cleric. The abuser is often an adult whom the child knows and trusts. When the abuser is someone close to the child, the child is likely to be torn on the one hand by allegiance to the abuser and, on the other hand, by fear, revulsion and the knowledge that what is happening is wrong. The betrayal of this trust makes the crime more abhorrent than it would be if no prior relationship existed between abuser and child. As with childhood incest, molestation or sexual harassment by an authority figure violates trust and respect. The victim, whatever his or her age, cannot give meaningful consent. The power differential is just too great.

Here, we consider two central questions. How does the all-too-common experience of CSA affect mental health during childhood and beyond? What can be done to help children heal from CSA?

#### **Effects on the child**

About half of children who are exposed to CSA will develop symptoms, such as depression, low self-esteem, conduct disorder and anxiety disorders like post-traumatic stress disorder (PTSD). On the other hand, almost half of children who are exposed to CSA do not appear to experience immediate symptoms (Kuehnle, 1998). What factors contribute to how CSA affects a child? The odds that CSA will produce clinically significant symptoms are increased when a perpetrator threatens the child, the child blames himself or herself or the family is unsupportive (Kuehnle, 1998). Negative outcomes are more pronounced when the CSA involves sexual intercourse (Nelson et al., 2002). Symptoms also appear to be more likely when the CSA started at an earlier age (Kaplow & Widom, 2007).

In adulthood, CSA is related to higher risk of many different psychological disorders when assessed in representative community samples (Afifi et al., 2008). We have seen in previous chapters that a history of CSA is common among adults experiencing many different psychological disorders — notably, dissociative identity disorder, PTSD, eating disorders, borderline personality disorder, major depressive disorder, sexual dysfunctions and substance abuse. CSA is also related to changes in the function of the HPA axis and the regulation of the stress hormone, cortisol, during adulthood (Stein, Yehuda, Koverola, & Hanna, 1997).

An issue in interpreting these correlations is that families in which abuse occurs are often experiencing a broad array of problems, such as substance dependence in one or both parents, which may be entangled with other genetic and environmental risks for psychopathology. As a result, it is hard to isolate whether CSA is genuinely the factor that heightens the risk for a clinical disorder — most children who are exposed to child abuse also experience other forms of early adversity (Green et al., 2010). Twin studies provide a way to disentangle these effects, particularly when one twin but not the other has been abused, because the twin who was not abused shares genetic, and at least some environmental, risk factors. In one study of almost 2000 twin pairs, adults with a history of CSA had substantially increased risk of depression, suicide, conduct disorder, alcohol dependence, social anxiety, rape and divorce compared to their non-abused twins (Nelson et al., 2002).

#### **Dealing with the problem**

When they suspect that something is awry, parents must raise the issue with their children; unfortunately, many adults are uncomfortable doing so. Physicians also need to be sensitive to signs of sexual abuse. Sexual and non-sexual forms of abuse are reportable offences; professionals who suspect abuse are required by law to report their suspicions to the police or child protective agencies. For a child, reporting sexual abuse can be extremely difficult. We tend to forget how helpless and dependent the

child feels, and it is difficult to imagine how frightening it would be to tell one's parents that one had been fondled by a brother or grandfather. Most cases of sexual abuse do not leave any physical evidence, such as torn vaginal tissue. Furthermore, there are no behavioural signs or emotional syndromes that unequivocally indicate that abuse has occurred (Kuehnle, 1998). Therefore, the child's own report is the primary source of information about whether CSA has occurred. The problem is that leading questions can produce some false reports. Great skill is required in questioning a child about possible sexual abuse to avoid biasing the youngster one way or the other. To reduce the stress on the child while protecting the rights of the accused adult, some jurisdictions use procedures such as videoed testimony, closed-courtroom trials, closed-circuit televised testimony and special coaching sessions to explain what to expect in the courtroom (Wolfe, 1990). Having the child play with anatomically correct dolls can be useful, but it should be only one part of an assessment because many non-abused children portray such dolls having sexual intercourse (Jampole & Weber, 1987). It is impossible to know what percentage of incest cases are not reported to the police, but it is safe to say that most are unreported, particularly if the offender is a family member (Gomes, Jardim, Taveira, Dinis-Oliveira, & Magalhaes, 2014).

Many children need treatment (Litrownik & Castillo-Canez, 2000). As with adult survivors of rape, PTSD can be a consequence. Many interventions are similar to those used for PTSD in adults; the emphasis is on exposure to memories of the trauma through discussion in a safe and supportive therapeutic atmosphere. It is also important for children to learn that healthy human sexuality is not about power and fear (McCarthy, 1986). As with rape, it is important to change the person's attribution of responsibility from 'I was bad' to 'he/she was bad'. Findings of a randomised controlled trial suggest that such treatments can help abused children find relief from their symptoms and can reduce their sense of shame (Cohen, Deblinger, Mannarino, & Steer, 2004)

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### QUESTIONS

1. Why do you think CSA is under-reported?
2. What could we do as a society to improve the reporting rates of CSA?

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About half of children who are exposed to CSA will develop symptoms, such as depression, low self-esteem, conduct disorder and anxiety disorders like post-traumatic stress disorder (PTSD).



Although the number of men who seem to experience a sexual attraction to children might seem disturbing (and perhaps even more disturbing for some of the men who experience the urges), the diagnosis of paedophilia is not made on the basis of sexual attraction alone. Paedophilic disorder is diagnosed *only* when adults act on their sexual urges towards children or when the urges reach the frequency or intensity to be distressing to the person or those close to them.

Incest is listed as a subtype of paedophilic disorder. **Incest** refers to sexual relations between close relatives for whom marriage is forbidden. It is most common between brother and sister, as depicted in the Game of Thrones series between Queen Cersai and her twin brother Ser Jaime Lannister. The next most common form, which is considered more pathological, is between father and daughter.

The taboo against incest is virtually universal in human societies (Ford & Beach, 1951), with the notable exception of Egyptian pharaohs, who could marry their sisters or other females of their immediate families. In Egypt, it was believed that the royal blood should not be contaminated by that of outsiders. The incest taboo makes sense according to present-day scientific knowledge. The offspring from a father–daughter or a brother–sister union have a greater probability of inheriting a pair of recessive genes, one from each parent. Many recessive genes have negative biological effects, such as serious birth defects. The incest taboo, then, has adaptive evolutionary significance.

Men who commit incest usually abuse their pubescent daughters, whereas men with non-incestual paedophilic disorder are usually interested in pre-pubertal children. Consistent with this difference in the age of victims, men who molest children within their families show greater penile arousal (as measured by penile plethysmography) to adult heterosexual cues than do men who molest unrelated children (Marshall, Barbaree, & Christophe, 1986).

## Voyeuristic disorder

The central feature of **voyeuristic disorder** is an intense and recurrent desire to obtain sexual gratification by watching unsuspecting others in a state of undress or having sexual relations. Voyeuristic fantasies are quite common in men, but as with the other paraphilic disorders, fantasies alone do not warrant a diagnosis. For some men with this disorder, voyeurism is their only sexual activity; for others, it is preferred but not absolutely essential for sexual arousal (Kaplan & Kreuger, 1997). Oftentimes, the looking, sometimes called peeping, helps the person become sexually aroused and is sometimes essential for arousal. People with voyeuristic disorder achieve orgasm by masturbation, either while watching or later while remembering the peeping. Sometimes the person with voyeuristic disorder fantasises about having sexual contact with the observed person, but it remains a fantasy; he or she seldom contacts the observed person. A true voyeur, almost always a man, does not find it particularly exciting to watch a woman who is undressing for his benefit. The element of risk seems important, for the voyeur is excited by the anticipation of how the woman would react if she knew he was watching.

### DSM-5

#### DSM-5 criteria for voyeuristic disorder

- People have voyeuristic disorder if, for at least six months, they experience recurrent and intense sexually arousing fantasies, urges or behaviours involving the observation of unsuspecting others who are naked, disrobing or engaged in sexual activity.
- Voyeuristic disorder is diagnosed if the person has acted on these urges with a non-consenting person or the urges and fantasies cause marked distress or interpersonal problems.

## Exhibitionistic disorder

The focus of sexual desire in **exhibitionistic disorder** is on exposing one's genitals to an unwilling stranger, sometimes a child. As with voyeuristic disorder, there is seldom an attempt to have other

contact with the stranger. Many exhibitionists masturbate during the exposure. In most cases, there is a desire to shock or embarrass the observer. The urge to expose seems overwhelming and virtually uncontrollable to the exhibitionist and is apparently triggered by anxiety and restlessness as well as by sexual arousal. In the tension of the moment, many describe symptoms of anxiety, including headaches, palpitations and derealisation. Because of the compulsive nature of the urge, the exposures may be repeated often and typically in the same place and at the same time of day. At the time of the act, the social and legal consequences are far from exhibitionists' minds (Stevenson & Jones, 1972). After exposing themselves, exhibitionists tend to flee and feel remorseful. In one study, persons diagnosed with exhibitionistic disorder reported that they had been arrested for only 1 out of every 150 incidents (Abel et al., 1987).

#### DSM-5

##### DSM-5 criteria for exhibitionistic disorder

- A person with exhibitionistic disorder will experience, for at least six months, recurrent, intense and sexually arousing fantasies, urges or behaviours involving showing one's genitals to an unsuspecting person.
- Exhibitionistic disorder is diagnosed if the person has acted on these urges to a non-consenting person or the urges and fantasies cause clinically significant distress or interpersonal problems.

## Frotteuristic disorder

The focus of sexual desire and urges in **frotteuristic disorder** is on touching an unsuspecting person. The person with this disorder may rub his penis against a woman's thighs or buttocks or fondle her breasts or genitals. These attacks typically occur in places such as a crowded bus or sidewalk that provide an easy means of escape. Most men who engage in frotteurism report doing so dozens of times (Abel et al., 1987). Frotteuristic disorder has not been studied extensively.

#### DSM-5

##### DSM-5 criteria for frotteuristic disorder

- Frotteuristic disorder is diagnosed if the person, for at least six months, experiences recurrent and intense and sexually arousing fantasies, urges or behaviours involving touching or rubbing against a non-consenting person.
- Frotteuristic disorder is diagnosed if the person has acted on these urges with a non-consenting person or the urges and fantasies cause clinically significant distress or problems.

## Sexual sadism and masochism disorders

The focus of desire in **sexual sadism disorder** is on inflicting pain or psychological suffering (such as humiliation) on another and the focus of desire in **sexual masochism disorder** is on being subjected to pain or humiliation. Some sadists achieve orgasm by inflicting pain and some masochists achieve orgasm by being subjected to pain. The manifestations of sexual masochism disorder are varied. Examples include physical bondage, blindfolding, spanking, whipping, electric shocks, cutting, humiliation (e.g., being urinated or defecated on, being forced to wear a collar and bark like a dog or being put on display naked) and taking the role of slave and submitting to orders and commands. Most sadists establish relationships with masochists to derive mutual sexual gratification. Although many people are able to take both dominant and submissive roles, masochists outnumber sadists.

## DSM-5

### DSM-5 criteria for sexual sadism disorder

- Sexual sadism disorder is diagnosed if, for at least six months, the person experiences recurrent, intense and sexually arousing fantasies, urges or behaviours involving the physical or psychological suffering of another person.
- Sexual sadism disorder is diagnosed if the symptoms cause clinically significant distress or impairment in functioning or the person has acted on these urges with a non-consenting person.

The release of books and movies such as *Fifty Shades of Grey* have seen more people try sadomasochistic sexual activity.



Sadistic and masochistic sexual behaviours have become more accepted over time: 5 to 10 percent of the population have tried some form of sadomasochistic activity, such as blindfolding or spanking one's partner (Baumeister & Butler, 1997). This has also been seen with the release of books and now movies such as *Fifty Shades of Grey* (2015) and its sequel *Fifty Shades Darker* (2017). In major cities, clubs cater to members seeking sadomasochistic partnerships. Most people who engage in sadomasochistic behaviours are relatively comfortable with their sexual practices and would not meet the diagnostic criteria requiring that the desires lead to distress or impairment (Spengler, 1977).

Because these disorders have become more common and are typically not related to impairment or distress, there was debate about whether these diagnoses should be retained in DSM-5 (Krueger, 2010b).

These diagnostic labels were retained because some sadistic and masochistic practices can be dangerous. One particularly dangerous form of masochism, called hypoxophilia (commonly known as erotic asphyxia or, if practised alone, known as auto-erotic asphyxia), can result in death or brain damage; it involves sexual arousal by restricting breathing, which can be achieved using a noose, a plastic bag or chest compression. More commonly, the diagnosis is applicable when the sadomasochistic urges and preferences lead to either personal or relationship distress.

#### RESEARCH EXAMPLE

##### **Michael Hutchence (1960–1997) — suicide or auto-erotic asphyxiation (AEA)?**

Some might remember Michael Hutchence as the lead singer in INXS — an Australian band with global popularity — or as a man who dated famous Australians. Others might remember the reports of how he took his life with a leather belt around his neck — found naked in a hotel room (Hand & Fife-Yeomans, 2008). To most, this sounds like suicide; however, to people who work in the area of abnormal psychology or sexology, the immediate thought is auto-erotic asphyxiation (AEA), as it has all the hallmarks of AEA — Hutchence was found naked, alone and with an instrument that cuts oxygen to the brain.

Typically news and even coroner reports will only state ‘fatal suicide’, rather than suspected AEA as the official result of death. In this case, Hutchence’s wife, Paula Yates, even attempted to overturn the coroner’s suicide verdict, suggesting that it was most likely AEA (BBC, 1999). Often misunderstood and not a common practice, AEA may result in about one death per one million (Danese, 1990). People engage in the practice of AEA to experience a heightened state of sexual pleasure, and repeat this until it becomes a fatal hazard of a dangerous sexual practice (Hall, 1994).

#### QUESTION

Can you think of any other sexual practices that could be dangerous, or even fatal?

There is also some concern that the diagnosis of sexual sadism disorder is rarely applied in clinical settings. In an unpublished review of over 500 million visits to psychiatrists, gynaecologists, urologists and other physicians, no doctor recorded a diagnosis of sexual sadism disorder (Narrow, 2008, cited in Krueger, 2010a). Doctors in clinical settings may not use the diagnosis even when symptoms are present because of worries over stigma. The diagnosis, then, seems to be applied almost entirely within forensic settings (Krueger, 2010a).

#### DSM-5

##### **DSM-5 criteria for sexual masochism disorder**

- Sexual masochism disorder is diagnosed if, for at least six months, the person has experienced recurrent, intense and sexually arousing fantasies, urges or behaviours involving the act of being humiliated, beaten, bound or made to suffer.
- Causes marked distress or impairment in functioning.

Both of these disorders are found in heterosexual and homosexual relationships. Surveys have found that 20 to 30 percent of the members of sadomasochistic clubs are female (Moser & Levitt, 1987) and it has been assumed that a similar gender ratio might be true of diagnoses of sexual sadism and masochism. Most sadists and masochists lead otherwise conventional lives and there is some evidence that they are above average in income and educational status (Moser & Levitt, 1987). Alcohol abuse is common among sadists (Allnutt, Bradford, Greenberg, & Curry, 1996).

## Aetiology of the paraphilic disorders

Here, we will discuss the aetiology of the paraphilic disorders, including the role of neurobiological factors, early abuse and psychological variables. Because many people do not want to volunteer to talk about their paraphilias, researchers have few opportunities to understand their causes. Indeed, most studies rely on very small samples. As an example, only four studies were published between 1990 and 2009 that included more than 25 participants diagnosed with fetishistic disorder (Kafka, 2010). Beyond the lack of research and the small sample sizes, most of the research focuses on men who are arrested for their sexual behaviour; very little is known about those whose sexual behaviour does not lead to arrest. Hence, much of this literature is most relevant for understanding sexual offenders, who represent a more severe subset of those with paraphilic disorders.

### Neurobiological factors

Because the overwhelming majority of people with paraphilic disorders are men, there has been speculation that androgens (hormones such as testosterone) play a role. Androgens regulate sexual desire and sexual desire appears to be atypically high among sexual offenders with paraphilic disorders (Kafka, 1997). Having said that, men with paraphilic disorders do not appear to have high levels of testosterone or other androgens (Thibaut, De La Barra, Gordon, Cosyns, & Bradford, 2010).

### Childhood sexual abuse

Across multiple studies of adult offenders, about two-thirds of sexual offenders reported a history of sexual abuse, a rate that is more than threefold higher than the rate among those charged with non-sexual offences (Jespersen, Lalumiere, & Seto, 2009). But this cannot be the whole story — several large-scale follow-up studies of boys with confirmed sexual abuse have shown that fewer than 5 percent were charged with any type of sexual offence as adults (Ogloff, Cutajar, Mann, & Mullen, 2012; Salter et al., 2003).

### Psychological factors

Psychological research tends to consider the immediate triggers of sexual behaviour and the more distal personality and cognitive risk factors for paraphilic disorders. We'll cover triggers, then the broader risk factors and then we will discuss specific models of paedophilia.

For some of the paraphilias, succumbing to the sexual urge can be thought of as an impulsive act, in which the person loses control over their behaviour. Alcohol decreases the ability to inhibit impulses and, accordingly, incidents of paedophilic disorder, voyeuristic disorder and exhibitionistic disorder often occur in the context of alcohol use. Others report that their sexual behaviours are most likely to happen in the context of negative moods, suggesting that sexual activity is being used as a means to escape from negative affect.

If the immediate act can be triggered by negative emotions and by a loss of impulse control, then it should not be surprising that personality traits relevant to those triggers are often observed in those with paraphilic disorder. People with paraphilic disorders do tend to show heightened impulsivity and poor emotional regulation (Ward & Beech, 2006).

Cognitive distortions and attitudes also play a role in the paraphilic disorders. Men who engage in paraphilias that involve non-consenting women may have hostile attitudes and a lack of empathy towards women. Others may have distortions in the ways they think about their sexual behaviour. For example, a voyeur may believe that a woman who left her blinds up while undressing wanted someone to look at her (Kaplan & Kreuger, 1997). A person with paedophilic disorder may believe that children want to have sex with adults (Marshall, 1997) or may think of themselves as innocently teaching a child about sex (Maletzky, 2002). Men who engage in rape may blame the woman, saying that she 'deserved a lesson' or that her dress was provocative. See focus on discovery 9.4 for a discussion of rape.

A separate line of work focuses on the psychological traits associated with paedophilia. On average, men with paedophilic disorder have a slightly lower IQ and higher rates of neurocognitive problems than the general population (Cantor, Blanchard, Robichaud, & Christensen, 2005; Suchy, Eastvold, Strassberg, & Franchow, 2014). Academic problems are common, as are other criminal behaviours (Becker & Hunter, 1997). Beyond the cognitive deficits related to paedophilia, it is important to understand that there may be more than one psychological pathway to paedophilia (Knight & King, 2012). Some paedophiles show an intense preoccupation with sex, a sense of emotional compatibility with children and a specific sexual preference for children. Other paedophiles demonstrate a general profile of elevated impulsivity and psychopathy compared to the general population (Mann, Hanson, & Thornton, 2010).

#### FOCUS ON DISCOVERY 9.4

##### The rapist

(Note: This is an example of a male rapist and a female victim. Please be mindful that a 'rapist' can be any gender and a 'victim' can also be any gender. The most commonly reported type of rape is that where a man is the rapist and a woman is the victim, hence the use of that example here.)

Is the rapist primarily someone who seeks the thrill of dominating and humiliating a woman through intimidation and assault? Is he an ordinarily unassertive man with a fragile ego who, feeling inadequate after disappointment and rejection in work or love, takes out his frustrations on an unwilling stranger? What types of characteristics distinguish rapists? Can treatment reduce the risk of recidivism?

##### Understanding the aetiology of rape

There is no DSM diagnosis to capture a tendency to rape. Several traits appear to be elevated among rapists. Sexually aggressive men tend to show antisocial and impulsive personality traits (Knight, 2010) and unusually high hostility towards women (Malamuth, 1998). More specific to sexual interactions, rapists appear to have a high sex drive (as measured by frequency of sexual outlets) (Kafka, 1997) and may also have cognitive distortions that lead them to minimise the consequences of their behaviour. Some rapists seem to have problems distinguishing friendliness from seductiveness and inaccurately reading cues from a woman indicating that she wants intimacies to cease (Malamuth & Brown, 1994). In one major community survey, men who had sexually assaulted women were 3.5 times more likely to report erectile dysfunction than those who had not committed sexual assaults (Laumann, et al., 1999). It is also likely that there may be different subgroups of rapists, who show different clusters of traits: some may show more prominent sadistic traits, some may show more hyper-sexuality and some may show more impulsive traits (Knight, 2010).

Data also suggests that exposure to violence may increase the likelihood of rape. That is, rapists are more likely than non-rapists to have been the victim of sexual and physical abuse (Knight & Sims-Knight, 2011). Even watching violence against women in films can lead men to view violence as more acceptable. In a controlled experiment, undergraduate men who stated that they regarded rape as unacceptable were aroused by video portrayals of rape if the woman was depicted as having an orgasm during the assault (Malamuth & Check, 1983). Since the time this study was conducted, eight experiments have been conducted in which men are asked to watch videos that contain either sexual activities with violence or sexual activities without violence. A meta-analysis of these studies suggested that after watching the videos that contained violence, men were significantly more likely to report that violence towards women was acceptable (Allen, D'Alessio, & Brezgel, 1995). This research suggests that rape may be encouraged by pornography that depicts women enjoying violent sexual relations and more broadly highlights the importance of social factors.

In 1944, Frank Loesser released the song 'Baby, It's Cold Outside', which has been adapted by numerous singers since. To name a few: Johnny Mercer and Margaret Whiting (1949), Dean Martin and Doris Day (1949), Lady Gaga and Joseph Gordon-Levitt (2013), Michael Bublé and Idina Menzel (2014) and Brett Eldredge and Meghan Trainor (2016). At first, this sounds like a cute and flirty song, but when you review the lyrics a bit closer it's clear the responses are about sexual coercion (at one point, the question is even asked: 'What's in this drink?'). The song can be viewed at: [www.youtube.com/watch?v=7MFJ7ie\\_yGU](https://www.youtube.com/watch?v=7MFJ7ie_yGU)

At its heart, the classic tale of *Beauty and the Beast* could be interpreted as a textbook story of abuse. In the Disney film version, the beautiful Belle must fall in love with the abusive and violent Beast in order to turn him back into a prince. But the cursed prince imprisons Belle inside the castle, effectively centring the plot of the movie around her Stockholm syndrome as she develops 'feelings' for the Beast.



#### Psychological treatment for rapists

Treatment programs for rapists rely on the general approaches we describe for paraphilic disorders: motivational strategies, a range of cognitive-behavioural techniques and pharmacological treatments. As with the research on treatment for paraphilic disorders, the evidence regarding the effectiveness of these approaches is remarkably slim. Only one study has randomly assigned men who are sexual offenders to cognitive-behavioural treatment or to no treatment (Marques, Wiederanders, Day, Nelson, & van Ommeren, 2005). Examination of the outcomes for the 24 rapists who took part in that study suggested that 20 percent of men in the treatment group committed an offence during the five-year follow-up period, compared to 29 percent in the no-treatment group. Although this may seem like a small gain from treatment, any gain is important with such a difficult problem.

#### QUESTIONS

1. What other treatment programs do you know of for rapists?
2. What do you believe could be done to increase the positive outcomes of psychological treatment for rapists?

## Treatments for the paraphilic disorders

Findings on the treatment of paraphilic disorders are hard to interpret for several reasons. Most of the available research on treatment focuses on men who have been charged with sexual offences and those who are court-ordered into treatment. Beyond the lack of representative samples, outcomes in this area are highly variable; perhaps this is not surprising given that researchers have varied in their focus on

adolescent or adult offenders or on the most or least severe offenders. In a meta-analysis of 12 studies of men arrested for sexual offences, those who received treatment of some form — either biological or psychological — were about a third less likely to have repeat offences than those who did not receive treatment (Hall, Hirschman, & Oliver, 1995). Caution is warranted in interpreting this finding, for two reasons. First, because many researchers consider it unethical to withhold treatment when the consequences of sexual offences are so severe, most studies have not randomly assigned people into control groups. Rather, outcomes for those who complete treatment are typically compared to outcomes for men who were not offered treatment or who refused treatment. Second, most studies have not included long-term follow-up, even though recidivism increases as the years go by (Maletzky, 2002). With these issues as background, we now describe cognitive-behavioural and biological treatments for the paraphilic disorders.

### **Strategies to enhance motivation**

Sex offenders often lack the motivation to change their illegal behaviour. They may deny their problem, minimise the seriousness of their problem and feel confident that they can control their behaviour without any professional assistance. Some blame the victim, even a child, for being overly seductive. Many refuse to take part in treatment and even among those who begin treatment, many will drop out. To enhance motivation for treatment, a therapist can bolster the client's hope that he can gain control over his urges through treatment, highlight the potential legal and other consequences of continued engagement in the same sexual behaviour and note that plethysmograph assessments will make it hard to 'fake' a recovery (Miller & Rollnick, 1991).

### **Cognitive-behavioural treatment**

In the earliest years of behavioural treatment, paraphilic disorders were narrowly viewed as attractions to inappropriate objects and activities. Looking to behavioural psychology for ways to reduce these attractions, researchers fixed on aversion therapy. Thus, a person with a boot fetish would be given a shock on the hands or a drug that produces nausea when looking at a boot and so on. In the form of aversion therapy called satiation, men are coached to pair their paraphilic fantasies with another aversive stimulus: masturbating for 55 minutes after orgasm (Kaplan & Krueger, 2012). Another variation of aversion therapy is covert sensitisation, in which the person imagines situations he finds inappropriately arousing while at the same time also imagines feeling sick or ashamed for feeling and acting this way. Studies of covert sensitisation have shown that it reduces deviant arousal, but little evidence is available that these techniques alone actually change the behaviour (Maletzky, 2000).

Cognitive procedures are often used to counter the distorted thinking of people with paraphilic disorders. For example, an exhibitionist might claim that the girls he exposes himself to are too young to be harmed by it. The therapist would counter this distortion by pointing out that the younger the victim, the worse the harm will be (Maletzky, 1997).

In general, cognitive and behavioural approaches have become more sophisticated and broader in scope since the 1960s, when the paraphilic disorders were addressed almost exclusively through aversive conditioning and cognitive interventions. Current approaches supplement these traditional approaches with techniques such as social skills training and sexual impulse control training (Maletzky, 2002). Training in empathy towards others is another increasingly common cognitive technique; teaching the sex offender to consider how his or her behaviour would affect someone else may lessen the tendency to engage in such activities. Relapse prevention, modelled after the work on substance abuse described in the chapter on substance use disorders, is also an important component of many broader treatment programs. A therapist who uses relapse prevention techniques would help a person identify situations and emotions that might trigger symptomatic behaviour. Interventions that combine cognitive interventions with behavioural interventions appear to be more successful than those that are strictly behavioural (Hall et al., 1995).

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Training in empathy towards others is another increasingly common cognitive technique.



As with the treatment literature, most of the available evidence for cognitive-behavioural interventions is based on studies with no adequate control group. Without a control group, it seemed that psychological interventions were helping relieve symptoms — many people reported diminished paraphilic symptoms after treatment (Maletzky, 2002). Unfortunately, when compared to a control group that did not receive treatment, cognitive-behavioural therapy did not appear to reduce legal recidivism (Marques et al., 2005). Although these findings are discouraging, this remains an important area for ongoing research.

### **Biological treatments**

A variety of biological interventions have been tried on sex offenders. Castration, or removal of the testes, was used a great deal until hormonal treatments (described next) became available (Wille & Boulanger, 1984). Surgical castration is not a common treatment today due to major ethical concerns.

On the other hand, several medications have been used to treat paraphilic disorders, particularly among sex offenders. Typically, these medications are used as a supplement to psychological treatment. Among men, sexual drive and functioning are regulated by androgens (such as testosterone). Hence, hormonal agents that reduce androgens have been used to treat paraphilic disorders, including medroxyprogesterone acetate (MPA, trade name Depo-Provera) and cyproterone acetate (CPA, Gyrostat). Randomised controlled trials show that these agents reduce arousal to deviant objects, as measured using the penile plethysmograph (Thibaut et al., 2010). Despite the promising findings, ethical issues are raised about the indefinite use of hormonal agents. Long-term use of hormonal agents is associated with a few negative side effects, including infertility, liver problems, osteoporosis and diabetes. Informed consent concerning these risks must be obtained and many patients will not agree to use these drugs long-term (Hill, Briken, Kraus, Strohm, & Berner, 2003).

Beyond drugs that influence hormones, SSRI antidepressants are commonly used. Although pre-post measures suggest that SSRIs reduce arousal to deviant objects, researchers have not conducted

randomised controlled trials of SSRIs compared to a control condition and so the quality of the research evidence is quite poor (Thibaut et al., 2010).

### **Balancing efforts to protect the public against civil liberties for those with paraphilias**

Most people are frightened by sexual offences, so balancing the protection of the public against civil liberties of sexual offenders is not easy. Issues arise in multiple aspects of the legal process and outcome, including how diagnoses of paraphilic disorders can influence institutionalisation, but also with laws concerning the public's 'right to know' when a sex offender is released.

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Balancing the protection of the public against civil liberties of sexual offenders is not easy.



In Australia, a post-sentence preventive detention order is available, which allows a person post-sentence to be detained indefinitely in prison to ensure adequate protection of the community if a person is deemed to have committed a serious sexual offence (WA & Qld, NSW) or a person who has committed an offence of a sexual nature (NSW; note, in Qld and WA, the preventive detention orders are indefinite whereas in NSW, a continuing detention order expired at the end of the period specified in the order or if not specified, within five years of the date it was made [McSherry et al., 2006]). Queensland, Western Australia and New South Wales have legislative schemes in place (McSherry, Keyzer, & Freiberg, 2006, pp. 29–30). The diagnosis of paraphilia, then, has significant implications for civil liberties: receipt of this diagnosis can lead to placement in a psychiatric facility. In this context, it has been argued that care should be taken to ensure that this set of diagnoses has validity (Wakefield, 2011).

Towards the end of a prison sentence across Australia, an order is made which requires the offender to be 'subject to ongoing supervision in the community on their release' (McSherry et al., 2006, p. 35). In addition to the post-sentence supervision, all Australian jurisdictions have a process for registering sex offenders to monitor their location upon release from prison (see table 9.5). The Australian National Child Offender Register (ANCOR) requires offenders to notify police of their address, places they frequent,

car registration and other personal details (Australian Criminal Intelligence Commission, 2016). Once on the register, offenders are prohibited from working in child-related employment.

**TABLE 9.5 Australian National Child (Sex) Offender Register (ANCOR)**

Jurisdiction	Act	Section	Register
ACT	<i>Crimes (Child Sex Offenders) Act 2005</i>	117–122	Child Sex Offenders Register
NSW	<i>Child Protection (Offenders Registration) Act 2000</i>	19–19B	Child Protection Register
NT	<i>Child Protection (Offenders Reporting And Registration) Act 2004</i>	64–68	Child Protection Offender Register
Qld	<i>Child Protection (Offender Reporting) Act 2004</i>	68–74	Child Protection Register
SA	<i>Child Sex Offenders Registration Act 2006</i>	60–63	Register of Child Sex Offenders
Tas	<i>Community Protection (Offenders Reporting) Act 2005</i>	42–45	Community Protection Offender Register
Vic	<i>Sex Offenders Registration Act 2004</i>	62–66D	Sex Offender Register
WA	<i>Community Protection (Offenders Reporting) Act 2004</i>	80–84	Community Protection Offender Register

*Source:* McSherry et al. (2006, p. 37).

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## SUMMARY

### 9.1 Describe the influence of culture and gender on sexual norms and summarise the sexual response cycle for men and women.

Sexuality is profoundly shaped by culture and experience, so it is important to be aware of subjective biases in thinking about diagnoses. Gender also shapes sexuality: for example, men report more frequent sexual thoughts and behaviours than do women.

Kaplan described four phases of the sexual response cycle: desire, excitement, orgasm and resolution. Over time, researchers have learned that the Kaplan phases do not fit the data for women in two ways: the desire and excitement phase may not be distinct stages and the Kaplan definition of the excitement phase may be overly biological.

### 9.2 Explain the symptoms, causes and treatments for sexual dysfunctions.

In the DSM-5, the sexual dysfunction disorders are divided as follows:

- sexual interest, desire and arousal disorders (female sexual interest/arousal disorder, male hypoactive sexual desire disorder and erectile disorder)
- orgasmic disorders (female orgasmic disorder, delayed ejaculation and premature ejaculation)
- sexual pain disorder (genito-pelvic pain/penetration disorder).

Although there are no good estimates of how many people meet full diagnostic criteria for sexual dysfunction disorders, in one major survey, 43 percent of women and 31 percent of men reported at least some symptoms of sexual dysfunction. People who experience one sexual dysfunction disorder often experience a comorbid sexual dysfunction disorder; for example, a man who is experiencing premature ejaculation may develop hypoactive sexual desire disorder.

Before diagnosing sexual dysfunction, it is important to rule out medical explanations for a symptom. The key aetiological variables involved in sexual dysfunctions appear to be previous sexual abuse, lack of sexual knowledge, relationship problems, psychological disorders such as depression or anxiety, and negative cognitions and attitudes about sexuality.

Cognitive-behavioural treatments for sexual dysfunction include couples therapy to improve the overall relationship and to build intimacy; techniques to reduce anxiety and increase knowledge; cognitive interventions; communication training; physical exercises to increase knowledge and awareness of the body; and the squeeze technique for the treatment of premature ejaculation. Medical treatments are increasingly popular, despite some criticism that such treatments do not address the interpersonal context in which sexual dysfunctions often emerge. PDE-5 inhibitors such as Viagra and Cialis are commonly used to treat erectile dysfunction and antidepressant drugs can be helpful in the treatment of premature ejaculation.

### 9.3 Explain the symptoms, causes and treatments for paraphilic disorders.

Paraphilic disorders are defined as a sexual attraction to an unusual sexual object or activity that lasts at least six months and causes significant distress or impairment. The DSM diagnostic criteria for paraphilic disorders are distinguished based on the object of sexual attraction. The major DSM-5 diagnoses of paraphilias include fetishistic disorder, paedophilic disorder, voyeuristic disorder, exhibitionistic disorder, frotteuristic disorder, sexual sadism disorder, sexual masochism disorder and transvestic disorder. (The last-named diagnosis was not discussed here.)

Researchers do not know the prevalence of these disorders. Few major studies have been done on the aetiology of paraphilic disorders and the available studies largely focus on sexual offenders. Neurobiological theory has focused on excessively high levels of male hormones (testosterone), but the theory has not received strong support. Sexual offenders do appear to have higher rates of sexual abuse than do other offenders, but very few children who are abused grow up to engage in sexual offences against others. Psychological theories focus on impulsivity, poor emotion regulation and distorted cognitions. Alcohol use and negative affect are often immediate triggers of inappropriate sexual behaviours. For paedophilia, there may be more than one pathway: some men seem sexually

preoccupied with children and experience a sense of emotional compatibility with children; other men have a profile of more general impulsive, psychopathic traits. Neurocognitive deficits and lower IQ are often observed in men diagnosed with paedophilic disorder.

There are a variety of treatment options for people with paraphilic disorders; however, the two most common approaches are cognitive-behavioural treatments such as aversive therapy, and biological treatments such as castration, which has more recently been replaced with hormonal treatment. Although there are mixed results, the most vital aspect of positive treatment outcomes is the motivation of the person seeking treatment, and therefore strategies to enhance motivation should be a priority.

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## KEY TERMS

**delayed ejaculation disorder** a disorder in men involving delay in reaching orgasm or inability to reach orgasm

**desire phase** the first stage of the sexual response cycle, characterised by sexual interest or desire, often associated with sexually arousing fantasies

**erectile disorder** a recurrent and persistent inability to attain an erection or maintain it until completion of sexual activity

**excitement phase** as applied by Masters and Johnson, the second stage of the sexual response cycle, characterised by pleasure associated with increased blood flow to the genitalia

**exhibitionistic disorder (exhibitionism)** marked preference for obtaining sexual gratification by exposing one's genitals to an unwilling observer

**female orgasmic disorder** a recurrent and persistent delay or absence of orgasm in a woman during sexual activity adequate in focus, intensity and duration; in many instances the woman may experience considerable sexual excitement

**female sexual interest/arousal disorder** a sexual dysfunction characterised by a loss of sexual interest (urges, fantasies or desires) or lack of physiological or subjective arousal to sexual cues

**fetishistic disorder** a paraphilic disorder that involves reliance on an inanimate object for sexual arousal

**frotteuristic disorder** a disorder in which the chief concern involves the sexually oriented touching of an unsuspecting person, typically in public places that provide an easy means of escape

**gender dysphoria** a disorder in which the person feels that their biological sex (physical body) does not match their emotional/psychological identity

**genito-pelvic pain/penetration disorder** a disorder in which the woman persistently experiences pain or vaginal muscle spasms when intercourse is attempted

**incest** sexual relations between close relatives, most often between daughter and father or between brother and sister

**male hypoactive sexual desire disorder** a sexual dysfunction disorder defined by absence of or deficiency in sexual fantasies and urges in men

**orgasm phase** the third stage of the sexual response cycle, characterised by a peak of sexual pleasure, generally including ejaculation in men and contraction of the outer vaginal walls in women

**paedophilic disorders** a disorder that is expressed by recurrent, intense sexually arousing fantasies, urges or behaviours involving pre-pubescent children or young adolescents

**paraphilia** an ongoing intense sexual fantasy, urge or behaviour involving an object, suffering or humiliation, or non-consenting partner, which causes distress to the person experiencing it

**paraphilic disorder** sexual attraction to unusual objects or unusual sexual activities that leads to social difficulties or distress

**penile plethysmograph** a device for detecting blood flow and thus for recording changes in the size of the penis

**premature (early) ejaculation** inability of the male to inhibit his orgasm long enough for mutually satisfying sexual relations

**resolution phase** the fourth and final stage of the sexual response cycle, characterised by an abatement of muscle tension, relaxation and a sense of wellbeing

**sexual dysfunctions** dysfunctions in which the appetitive or psychophysiological changes of the normal sexual response cycle are inhibited

**sexual masochism disorder** when a person is sexually aroused by personally being made to suffer physical pain and/or humiliation by being bound and beaten

**sexual response cycle** the general pattern of sexual physical processes and feelings, made up of four phases: desire, excitement, orgasm and resolution

**sexual sadism disorder** a paraphilic disorder defined by a marked preference for obtaining or increasing sexual gratification by inflicting pain or humiliation on another person

**spectator role** as applied by Masters and Johnson, a pattern of behaviour in which the individual's focus on and concern with sexual performance causes him or her to be an observer rather than a participant and thus impedes natural sexual responses

**vaginal plethysmograph** a device for measuring physiological signs of sexual arousal in women; the device is shaped like a tampon and is inserted into the vagina to measure increases in blood flow

**voyeuristic disorder** a disorder defined by a marked preference for obtaining sexual gratification by watching others in a state of undress or having sexual relations

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## WEBSITES

1. 'Call Me Cate: From a political strategist to a high profile transgender' is an episode of the ABC documentary series *Australian Story* on ABC Online. ([www.abc.net.au/austory/specials/callmecate/default.htm](http://www.abc.net.au/austory/specials/callmecate/default.htm))
2. 'Christopher Ryan: Are we designed to be sexual omnivores?' is a TED Talk that provides a historical overview of sex and relationships with some humour included. ([www.ted.com/talks/christopher\\_ryan\\_are\\_we\\_designed\\_to\\_be\\_sexual\\_omnivores#t-523068](http://www.ted.com/talks/christopher_ryan_are_we_designed_to_be_sexual_omnivores#t-523068))
3. Criminology Research Council Report is a detailed report completed by Monash University concerning preventative detention for 'dangerous' offenders in Australia. ([www.criminologyresearchcouncil.gov.au/reports/200405-03.pdf](http://www.criminologyresearchcouncil.gov.au/reports/200405-03.pdf))

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## REFERENCES

- Abel, G. G., Becker, J. V., Mittelman, M., Cunningham-Rathner, J., Rouleau, J. L., & Murphy, W. D. (1987). Self-reported sex crimes of nonincarcerated paraphiliacs. *Journal of Interpersonal Violence*, 2, 3–25.
- Afifi, T. O., Enns, M. W., Cox, B. J., Asmundson, G. J. G., Stein, M. B., & Sareen, J. (2008). Population attributable fractions of psychiatric disorders and suicide ideation and attempts associated with adverse childhood experiences. *American Journal of Public Health*, 98, 946–952.
- Allen, M., D'Alessio, D., & Brezgel, K. (1995). A meta-analysis summarizing the effects of pornography: II. aggression after exposure. *Human Communication Research*, 22, 258–283.
- Allnutt, S. H., Bradford, J. M., Greenberg, D. M., & Curry, S. (1996). Co-morbidity of alcoholism and the paraphilias. *Journal of Forensic Science*, 41, 234–239.
- Althof, S. E. (2014). Treatment of premature ejaculation: Psychotherapy, pharmacotherapy, and combined therapy. In Y. M. Binik & K. S. K. Hall (Eds.), *Principles and practice of sex therapy* (5 ed., pp. 112–137). New York: Guilford Press.
- Althof, S. E., Abdo, C. H., Dean, J., Hackett, G., McCabe, M., McMahon, C. G., et al. (2010). International Society for Sexual Medicine's guidelines for the diagnosis and treatment of premature ejaculation. *Journal of Sexual Medicine*, 7, 2947–2969.
- American Psychiatric Association. (2013a). *Diagnostic and statistical manual of mental disorders* (5th ed.). Washington, DC: Author.
- American Psychiatric Association. (2013b). *Fact sheet: Gender dysphoria*. Washington, DC: Author.
- Andersen, B. L., Cyranowski, J. M., & Aarestad, S. (2000). Beyond artificial, sex-linked distinctions to conceptualize female sexuality: Comment on Baumeister (2000). *Psychological Bulletin*, 126(3), 380–384.
- Andersen, B. L., Cyranowski, J. M., & Espindle, D. (1999). Men's sexual self-schema. *Journal of Personality and Social Psychology*, 76, 645–661.

- Angst, J. (1998). Sexual problems in healthy and depressed persons. *International Clinical Psychopharmacology*, 13, S1–S4.
- Australian Criminal Intelligence Commission (2016, June 24). National Child Offender System. Retrieved from [www.acic.gov.au/our-services/child-protection/national-child-offender-system](http://www.acic.gov.au/our-services/child-protection/national-child-offender-system)
- Australian Federation of AIDS Organisations, (2016). *HIV statistics in Australia*. Retrieved from [www.hivmediaguide.org.au/hiv-in-australia/hiv-statistics-australia/](http://www.hivmediaguide.org.au/hiv-in-australia/hiv-statistics-australia/)
- Bach, A. K., Wincze, J. P., & Barlow, D. H. (2001). Sexual dysfunction. In D. H. Barlow (Ed.), *Clinical handbook of psychological disorders* (pp. 562–608). New York: Guilford Press.
- Bancroft, J., Loftus, J., & Long, J. S. (2003). Distress about sex: A national survey of women in heterosexual relationships. *Archives of Sexual Behavior*, 32, 193–208.
- Bartlik, B., & Goldberg, J. (2000). Female sexual arousal disorder. In S. R. Lieblum & R. C. Rosen (Eds.), *Principles and practice of sex therapy* (3rd ed., pp. 85–117). New York: Guilford Press.
- Basson, R., Althof, S. A., Davis, S., Fugl-Meyer, K., Goldstein, I., Leiblum, S., et al. (2004). Summary of the recommendations on sexual dysfunctions in women. *Journal of Sexual Medicine*, 1, 24–34.
- Basson, R., Brotto, L. A., Laan, E., Redmond, G., & Utian, W. H. (2005). Assessment and management of women's sexual dysfunctions: Problematic desire and arousal. *Journal of Sexual Medicine*, 2, 291–300.
- Baumeister, R. F. (2000). Gender differences in erotic plasticity: The female sex drive as socially flexible and responsive. *Psychological Bulletin*, 126(3), 347–374.
- Baumeister, R. F., & Butler, J. L. (1997). Sexual masochism: Deviance without pathology. In D. R. Laws & W. O'Donohue (Eds.), *Sexual deviance* (pp. 225–239). New York: Guilford Press.
- Baumeister, R. F., Catanese, K. R., & Vohs, K. (2001). Is there a gender difference in strength of sex drive? Theoretical views, conceptual distinctions, and a review of relevant evidence. *Personality and Social Psychology Review*, 5(3), 242–273.
- BBC News (BBC). (1999, August 10). Paula challenges Hutchence verdict. Retrieved 9 December 2010.
- Beck, J. G., & Bozman, A. (1995). Gender differences in sexual desire: The effects of anger and anxiety. *Archives of Sexual Behavior*, 24, 595–612.
- Becker, J. V., & Hunter, J. A. (1997). Understanding and treating child and adolescent sexual offenders. In T. H. Ollendick & R. J. Prinz (Eds.), *Advances in clinical child psychology* (pp. 177–196). New York: Plenum.
- Bhugra, D., Popelyuk, D., & McMullen, I. (2010). Paraphilias across cultures: contexts and controversies. *Journal of Sex Research*, 47, 242–256.
- Binik, Y. M. (2010). The DSM diagnostic criteria for vaginismus. *Archives of Sexual Behavior*, 39, 278–291.
- Birnbaum, G. E., Reis, H. T., Mikulincer, M., Gillath, O., & Orpaz, A. (2006). When sex is more than just sex: Attachment orientations, sexual experience, and relationship quality. *Journal of Personality and Social Psychology*, 91, 929–943.
- Blanchard, R. (2010). The specificity of victim count as a diagnostic indicator of pedohebephilia. *Archives of sexual behavior*, 39, 1245–1252.
- Brotto, L. A., & Luria, M. (2014). Sexual interest/arousal disorder in women. In Y. M. Binik & K. S. K. Hall (Eds.), *Principles and practice of sex therapy* (5th ed., pp. 17–41). New York: Guilford Press.
- Burri, A., & Spector, T. (2011). Recent and lifelong sexual dysfunction in a female UK population sample: Prevalence and risk factors. *Journal of Sexual Medicine*, 8, 2420–2430.
- Buvat, J., Maggi, M., Gooren, L., Guay, A. T., Kaufman, J., Morgentaler, A., et al. (2010). Endocrine aspects of male sexual dysfunctions. *Journal of Sexual Medicine*, 7, 1627–1656.
- Cantor, J. M., Blanchard, R., Robichaud, L. K., & Christensen, B. K. (2005). Quantitative reanalysis of aggregate data on IQ in sexual offenders. *Psychological Bulletin*, 131, 555–568.
- Carvalho, A. A., Brotto, L. A., & Leal, I. (2010). Women's motivations for sex: Exploring the Diagnostic and Statistical Manual, fourth edition, text revision criteria for hypoactive sexual desire and female sexual arousal disorders. *Journal of Sexual Medicine*, 7, 1454–1463.
- Carvalho, J., & Nobre, P. (2010). Biopsychosocial determinants of men's sexual desire: Testing an integrative model. *Journal of Sexual Medicine*, 8, 754–763.
- Chivers, M. L., Seto, M. C., Lalumière, M. L., Laan, E., & Grimbos, T. (2010). Agreement of self-reported and genital measures of sexual arousal in men and women: A meta-analysis. *Archives of Sexual Behavior*, 39, 5–56.
- Clark, A. W., Thompson, D. G., Graham, D., & Cooper, J. M. (2014). Engineering DNA binding sites to assemble and tune plasmonic nanostructures. *Advanced materials*, 26, 4286–4292.
- Cohen, J. A., Deblinger, E., Mannarino, A. P., & Steer, R. (2004). A multi-site, randomized controlled trial for children with abuse-related PTSD symptoms. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 393–402.
- Danese, R. (1990, Jan 22). Sexual ritual courts death, expert says. *The Windsor Star*. Retrieved from <https://search-proquest-com.dbgw.lis.curtin.edu.au/docview/253848236?accountid=10382>
- Dawson, S. J., Bannerman, B. A., & Lalumière, M. L. (2016). Paraphilic interests: An examination of sex differences in a nonclinical sample. *Sexual Abuse: A Journal of Research and Treatment*, 28(1), 20–45. doi:<http://dx.doi.org/dbgw.lis.curtin.edu.au/10.1177/1079063214525645>
- De Cuypere, G., Tsjoen, G., Beerten, R., Selvaggi, G., De Sutter, P., Hoebeke, P., et al. (2005). Sexual and physical health after sex reassignment surgery. *Archives of Sexual Behavior*, 34, 679–690.

- de Visser, R. O., Badcock, P. B., Simpson, J. M., Grulich, A. E., Smith, A. M. A., Richters J., Rissel, C. (2014). Attitudes toward sex and relationships: the Second Australian Study of Health and Relationships. *Sexual Health*, 11, 397–405.
- Derogatis, L. R., & Burnett, A. L. (2008). The epidemiology of sexual dysfunctions. *Journal of Sexual Medicine*, 5, 289–300.
- Eardley, I., Donatucci, C., Corbin, J., El-Meliegy, A., Hatzimouratidis, K., McVary, K., et al. (2010). Pharmacotherapy for erectile dysfunction. *Journal of Sexual Medicine*, 7, 524–540.
- Emmelkamp, P. M. G. (2004). Behavior therapy with adults. In M. J. Lambert (Ed.), *Bergin and Garfield's handbook of psychotherapy and behavior change* (5th ed., pp. 393–446). New York: John Wiley & Sons.
- Fink, H. A., Mac Donald, R., Rutks, I. R., Nelson, D. B., & Wilt, T. J. (2002). Sildenafil for male erectile dysfunction: A systematic review and meta-analysis. *Archives of Internal Medicine*, 162, 1349–1360.
- Flood, A. (2015, June 23). Fifty Shades of Grey sequel breaks sales records. *The Guardian*. Retrieved from [www.theguardian.com/books/2015/jun/23/fifty-shades-of-grey-sequel-breaks-sales-records](http://www.theguardian.com/books/2015/jun/23/fifty-shades-of-grey-sequel-breaks-sales-records)
- Forbes, M. K., & Schniering, C. A. (2013). Are sexual problems a form of internalizing psychopathology? A structural equation modeling analysis. *Archives of Sexual Behavior*, 42, 23–34.
- Ford, C. S., & Beach, F. A. (1951). *Patterns of sexual behavior*. New York: Harper and Brothers.
- Frühhauf, S., Gerger, H., Schmidt, H. M., Munder, T., & Barth, J. (2013). Efficacy of psychological interventions for sexual dysfunction: A systematic review and meta-analysis. *Archives of Sexual Behavior*, 42, 915–933.
- Gomes, V., Jardim, P., Taveira, F., Dinis-Oliveira, R. J., & Magalhães, T. (2014). Alleged biological father incest: A forensic approach. *Journal of Forensic Sciences*, 59(1), 255–259.
- Goode, S. D. (2010). *Understanding and addressing adult sexual attraction to children: A study of paedophiles in contemporary society*. London: Routledge.
- Graham, C. A. (2010). The DSM diagnostic criteria for female sexual arousal disorder. *Archives of Sexual Behavior*, 39, 240–255.
- Graham, C. A. (2014). Orgasm disorders in women. In Y. M. Binik & K. S. K. Hall (Eds.), *Principles and practice of sex therapy* (5th ed., pp. 89–111). New York: Guilford Press.
- Green, J. G., McLaughlin, K. A., Berglund, P. A., Gruber, M. J., Sampson, N. A., Zaslavsky, A. M., et al. (2010). Childhood adversities and adult psychiatric disorders in the National Comorbidity Survey Replication: Associations with first onset of DSM-IV Disorders. *Archives of General Psychiatry*, 67, 113–123.
- Green, R., & Fleming, D. T. (1990). Transsexual surgery follow-up: Status in the 1990s. In J. Bancroft, C. Davis & D. Weinstein (Eds.), *Annual Review of Sex Research* (pp. 163–174).
- Hall, C. (1994, February 9). Fatal hazards of a dangerous sexual practice. *The Independent*. Retrieved from <https://search-proquest-com.dbgw.lis.curtin.edu.au/docview/313060329?accountid=10382>
- Hall, G. C., Hirschman, R., & Oliver, L. L. (1995). Sexual arousal and arousability to pedophilic stimuli in a community sample of normal men. *Behavior Therapy*, 26, 681–694.
- Hand, D. & Fife-Yeomans, J. (2008/2004). *The coroner: Investigating sudden death*. Sydney, NSW: Allen & Unwin.
- Hanson, R. K., & Bussiere, M. T. (1998). Predicting relapse: A meta-analysis of sexual offender recidivism studies. *Journal of Consulting and Clinical Psychology*, 66, 348–362.
- Hanson, R. K., & Harris, A. J. R. (1997). Voyeurism: Assessment and treatment. In D. R. Laws & W. O'Donohue (Eds.), *Sexual deviance* (pp. 311–331). New York: Guilford Press.
- Herbenick, D., Reece, M., Schick, V., Sanders, S. A., Dodge, B., & Fortenberry, J. D. (2010b). An event-level analysis of the sexual characteristics and composition among adults ages 18 to 59: Results from a national probability sample in the United States. *Journal of Sexual Medicine*, 7 Suppl 5, 346–361.
- Herbenick, D., Reece, M., Schick, V., Sanders, S. A., Dodge, B., & Fortenberry, J. D. (2010a). Sexual behaviors, relationships, and perceived health status among adult women in the United States: Results from a national probability sample. *Journal of Sexual Medicine*, 7 Suppl 5, 277–290.
- Herd, G. (1999). *Sambia sexual culture: Essays from the field*. Chicago: University of Chicago Press.
- Hill, A., Briken, P., Kraus, C., Strohm, K., & Berner, W. (2003). Differential pharmacological treatment of paraphilias and sex offenders. *International Journal of Offender Therapy and Comparative Criminology*, 47, 407–421.
- Jampole, L., & Weber, M. K. (1987). An assessment of the behavior of sexually abused and nonsexually abused children with anatomically correct dolls. *Child Abuse and Neglect*, 11, 187–192.
- Jespersen, A. F., Lalumière, M. L., & Seto, M. C. (2009). Sexual abuse history among adult sex offenders and non-sex offenders: a meta-analysis. *Child Abuse and Neglect*, 33, 179–192.
- Kafka, M. P. (1997). Hypersexual desire in males: An operational definition and clinical implications for males with paraphilias and paraphilia-related disorders. *Archives of Sexual Behavior*, 26(5), 505–526.
- Kafka, M. P., & Hennen, J. (2002). A DSM-IV Axis I comorbidity study of males ( $n = 120$ ) with paraphilias and paraphilia-related disorders. *Sexual Abuse: A Journal of Research and Treatment*, 14, 349–366.
- Kafka, M. P. (2010). The DSM diagnostic criteria for fetishism. *Archives of Sexual Behavior*, 39, 357–362.
- Kaplan, H. S. (1974). *The new sex therapy*. New York: Brunner/Mazel.
- Kaplan, H. S. (1997). Sexual desire disorders (hypoactive sexual desire and sexual aversion). In G. O. Gabbard & S. D. Atkinson (Eds.), *Synopsis of treatments of psychiatric disorders* (2nd ed., pp. 771–780). Washington, DC: American Psychiatric Press.

- Kaplan, M. S., & Kreuger, R. B. (1997). Voyeurism: Psychopathology and theory. In D. R. Laws & W. O'Donohue (Eds.), *Sexual deviance* (pp. 297–310). New York: Guilford Press.
- Kaplan, M. S., & Krueger, R. B. (2012). Cognitive-behavioral treatment of the paraphilias. *Israel Journal of Psychiatry Related Sciences*, 49, 291–296.
- Kaplow, J. B., & Widom, C. S. (2007). Age of onset of child maltreatment predicts long-term mental health outcomes. *Journal of Abnormal Psychology*, 116, 176–187.
- Karney, B. R., & Bradbury, T. N. (1995). The longitudinal course of marital quality and stability: A review of theory, method, and research. *Psychological Bulletin*, 118, 3–34.
- Kinsey, A. C., Pomeroy, W. B., & Martin, C. E. (1948). *Sexual behavior in the human male*. Philadelphia: Saunders.
- Kirby Institute. (2017). *Number of HIV notification, 1984–2015, by age group*. University of New South Wales. Retrieved from <https://data.kirby.unsw.edu.au/HIV>
- Kirby Institute. (November 14, 2016). *2016 Annual Surveillance Report of HIV, viral hepatitis, STIs*. University of New South Wales. Retrieved from <http://kirby.unsw.edu.au/surveillance/2016-annual-surveillance-report-hiv-viral-hepatitis-stis>
- Kleinplatz, P. J. (2014). The paraphilias: An experiential approach to "dangerous" desires. In Y. M. Binik & K. S. K. Hall (Eds.), *Principles and practice of sex therapy* (5th ed.). New York: Guilford Press.
- Knight, R. A. (2010). Typologies for rapists: The generation of a new structural model. In A. Schlink (Ed.), *The sexual predator* (Vol. 4, pp. 17–1–17–8) New York, NY: Civic Research Institute.
- Knight, R. A., & King, M. (2012). Typologies for child molesters: The generation of a new structural model. In B. K. Schwartz (Ed.), *The sexual offender*. Kingston, NJ: Civic Research Institute.
- Knight, R. A., & Sims-Knight, J. (2011). Risk factors for sexual violence. In J. White, M. Koss & A. E. Kazdin (Eds.), *Violence against women and children, Volume 1: Mapping the terrain*. Washington DC: American Psychological Association.
- Krueger, R. B. (2010a). The DSM diagnostic criteria for sexual masochism. *Archives of Sexual Behavior*, 39, 346–356.
- Krueger, R. B. (2010b). The DSM diagnostic criteria for sexual sadism. *Archives of Sexual Behavior*, 39, 325–345.
- Kuehnle, K. (1998). Child sexual abuse evaluations: The scientist-practitioner model. *Behavioral Sciences and the Law*, 16, 5–20.
- Laan, E., Everaerd, W., & Both, S. (2005). Female sexual arousal. In R. Balon & R. T. Segraves (Eds.), *Handbook of sexual dysfunctions and paraphilias*. Boca Raton, FL: Taylor and Francis Group.
- Långström, N. (2010). The DSM diagnostic criteria for exhibitionism, voyeurism, and frotteurism. *Archives of Sexual Behavior*, 39, 317–324.
- Långström, N., & Seto, M. C. (2006). Exhibitionistic and voyeuristic behavior in a Swedish national population survey. *Archives of Sexual Behavior*, 35, 427–435.
- Laumann, E. O., Nicolosi, A., Glasser, D. B., Paik, A., Gingell, C., Moreira, E., et al. (2005). Sexual problems among women and men aged 40–80 years: Prevalence and correlates identified in the global study of sexual attitudes and behaviors. *International Journal of Impotence Research*, 17, 39–57.
- Laumann, E. O., Paik, A., & Rosen, R. C. (1999). Sexual dysfunction in the United States: Prevalence and predictors. *Journal of the American Medical Association*, 281, 537–544.
- Leiblum, S. R. (1997). Sexual pain disorders. In G. O. Gabbard & S. D. Atkinson (Eds.), *Synopsis of treatments of psychiatric disorders* (2nd ed., pp. 805–810). Washington, DC: American Psychiatric Press.
- Lief, H. I., & Hubschman, L. (1993). Orgasm in the postoperative transsexual. *Archives of Sexual Behavior*, 22, 145–155.
- Lippa, R. A. (2009). Sex differences in sex drive, sociosexuality and height across 53 nations: Testing evolutionary and social structural theories. *Archives of Sexual Behavior*, 38(5), 631–51.
- Litrownik, A. F., & Castillo-Canez, I. (2000). Childhood maltreatment: Treatment of abuse and incest survivors. In C. R. Snyder & R. E. Ingram (Eds.), *Handbook of psychological change* (pp. 520–545). New York: John Wiley & Sons.
- LoPiccolo, J., & Lobitz, W. C. (1972). The role of masturbaton in the treatment of orgasmic dysfunction. *Archives of Sexual Behavior*, 2, 163–171.
- Malamuth, N. M. (1998). An evolutionary-based model integrating research on the characteristics of sexually coercive men. In J. G. A. a. D. Belanger (Ed.), *Advances in psychological science, Volume 1: Social, personal, and cultural aspects* (pp. 151–184). Hove, UK: Psychology Press/Erlbaum.
- Malamuth, N. M., & Check, J. V. P. (1983). Sexual arousal to rape depictions: Individual differences. *Journal of Abnormal Psychology*, 92, 55–67.
- Malamuth, N. M., & Brown, L. M. (1994). Sexually aggressive men's perceptions of women's communications: Testing three explanations. *Journal of Personality and Social Psychology*, 67, 699–712.
- Maletzky, B. M. (1997). Exhibitionism: Assessment and treatment. In D. R. Laws & W. O'Donohue (Eds.), *Sexual deviance* (pp. 40–74). New York: Guilford Press.
- Maletzky, B. M. (2000). Exhibitionism. In M. Hersen & M. Biaggio (Eds.), *Effective brief therapy: A clinician's guide* (pp. 235–257). New York: Plenum.
- Maletzky, B. M. (2002). The paraphilias: research and treatment. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work* (pp. 525–558). New York: Oxford University Press.
- Mann, R. E., Hanson, R. K., & Thornton, D. (2010). Assessing risk for sexual recidivism: Some proposals on the nature of psychologically meaningful risk factors. *Sexual Abuse: A Journal of Research and Treatment*, 22, 191–217.

- Marques, J. K., Wiederanders, M., Day, D. M., Nelson, C., & van Ommeren, A. (2005). Effects of a relapse prevention program on sexual recidivism: Final results from California's Sex Offender Treatment and Evaluation Project (SOTEP). *Sexual Abuse: A Journal of Research and Treatment*, 17, 79–107.
- Marshall, W. L. (1997). Pedophilia: Psychopathology and theory. In D. R. Laws & W. O'Donohue (Eds.), *Sexual deviance* (pp. 152–174). New York: Guilford Press.
- Marshall, W. L., Barbaree, H. E., & Christophe, D. (1986). Sexual offenders against female children: Sexual preferences for age of victims and type of behaviour. *Canadian Journal of Behavioural Science*, 18, 424–439.
- Masters, W. H., & Johnson, V. E. (1966). *Human sexual response*. Boston: Little, Brown.
- Masters, W. H., & Johnson, V. E. (1970). *Human sexual inadequacy*. Boston: Little, Brown.
- McCarthy, B. W. (1986). A cognitive-behavioral approach to understanding and treating sexual trauma. *Journal of Sex and Marital Therapy*, 12, 322–329.
- McGregor, C. (April 11, 2015). Being transgender: The secret I kept for 50 years. *Australian Women's Weekly*.
- McSherry, B., Keyzer, P., & Freiberg, A. (December 2006). Preventive detention for 'dangerous' offenders in Australia: A critical analysis and proposals for policy development. Report to the Criminology Research Council. Retrieved from: [www.criminologyresearchcouncil.gov.au/reports/200405-03.pdf](http://www.criminologyresearchcouncil.gov.au/reports/200405-03.pdf)
- Meana, M., Binik, I., Khalife, S., & Cohen, D. (1998). Affect and marital adjustment in women's ratings of dyspareunia pain. *Canadian Journal of Psychiatry*, 43, 381–385.
- Melnik, T., Soares, B. G. O., & Nasello, A. G. (2008). The effectiveness of psychological interventions for the treatment of erectile dysfunction: Systematic review and meta-analysis, including comparisons to sildenafil treatment, intracavernosal injection and vacuum devices. *Journal of Sexual Medicine*, 5(11), 2562–2574.
- Melnik, T., Soares, B. G. O., & Nasello, A. G. (2008). The effectiveness of psychological interventions for the treatment of erectile dysfunction: Systematic review and meta-analysis, including comparisons to sildenafil treatment, intracavernosal injection, and vacuum devices. *Journal of Sexual Medicine*, 5, 2562–2574.
- Mercer, C. H., Fenton, K. A., Johnson, A. M., Wellings, K., Macdowall, W., McManus, S., et al. (2003). Sexual function problems and help seeking behaviour in Britain: national probability sample survey. *British Medical Journal*, 327, 426–427.
- Meston, C. M., & Buss, D. (2009). *Why women have sex: Women reveal the truth about their sex lives, from adventure to revenge (and everything in between)*. New York: St. Martin's Press.
- Meston, C. M., & Gorzalka, B. B. (1995). The effects of sympathetic activation on physiological and subjective sexual arousal in women. *Behaviour Research and Therapy*, 33, 651–664.
- Miller, K. (n.d.). *Paedophilia: Policy and prevention. Detection and reporting of paedophilia: A law enforcement perspective*. Canberra: Australian Institute of Criminology. Retrieved from [www.aic.gov.au/media\\_library/conferences/paedophilia/miller.pdf](http://www.aic.gov.au/media_library/conferences/paedophilia/miller.pdf)
- Miller, W. R., & Rollnick, S. (Eds.). (1991). *Motivational interviewing: Preparing people to change addictive behavior*. New York: Guilford Press.
- Morenz, B., & Becker, J. V. (1995). The treatment of youthful sexual offenders. *Applied and Preventive Psychology*, 4, 247–256.
- Morokoff, P. J., & Gilliland, R. (1993). Stress, sexual functioning, and marital satisfaction. *Journal of Sex Research*, 30, 43–53.
- Moser, C., & Levitt, E. E. (1987). An exploratory descriptive study of a sadomasochistically oriented sample. *Journal of Sex Research*, 23, 322–337.
- Muehlenhard, C. L., & Shippee, S. K. (2010). Men's and women's reports of pretending orgasm. *Journal of Sex Research*, 47, 552–567.
- Nagaraj, A. K., Pai, N. B., Rao, S., Rao, T. S., & Goyal, N. (2009). Biology of sexual dysfunction. *Online Journal of Health and Allied Sciences*, 8(1), 1–7. Retrieved from <http://ro.uow.edu.au/medpapers/20>
- Nelson, E. C., Heath, A. C., Madden, P. A. F., Cooper, M. L., Dinwiddie, S. H., Bucholz, K. K., et al. (2002). Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: Results from a twin study. *Archives of General Psychiatry*, 59, 139–145.
- Nicholls, L. (2008). Putting the New View Classification Scheme to an empirical test. *Feminism and Psychology*, 18, 515–526.
- Nobre, P. J., & Pinto-Gouveia, J. (2008). Cognitions, emotions, and sexual response: Analysis of the relationship among automatic thoughts, emotional responses, and sexual arousal. *Archives of Sexual Behavior*, 37, 652–661.
- O'Donohue, W., Dopke, C. A., & Swingen, D. N. (1997). Psychotherapy for female sexual dysfunction: A review. *Clinical Psychology Review*, 17, 537–566.
- O'Grady, R. (2001). Eradicating pedophilia toward the humanization of society. *Journal of International Affairs*, 55(1), 123–140.
- Ogloff, J. R. P., Cutajar, M. C., Mann, E., & Mullen, P. (2012). Child sexual abuse and subsequent offending and victimisation: A 45 year follow-up study. *Trends and Issues in Crime and Criminal Justice*, 440.
- Petersen, J. L., & Hyde, J. S. (2010). A meta-analytic review of research on gender differences in sexuality, 1993–2007. *Psychological Bulletin*, 136(1), 21. Retrieved from <https://search-proquest-com.dbgw.lis.curtin.edu.au/docview/203479828?accountid=10382>
- Polonsky, D. C. (2000). Premature ejaculation. In R. C. Rosen & S. R. Leiblum (Eds.), *Principles and practice of sex therapy* (3rd ed., pp. 305–332). New York: Guilford Press.
- PR Newswire (2013). Erectile dysfunction drugs market is expected to reach USD \$3.4 billion globally in 2019: Transparency Market Research. Albany, NY. Retrieved from: [www.prnewswire.com/news-releases/erectile-dysfunction-drugs-market-is-expected-to-reach-usd-34-billion-globally-in-2019-transparency-market-research-228593931.html](http://www.prnewswire.com/news-releases/erectile-dysfunction-drugs-market-is-expected-to-reach-usd-34-billion-globally-in-2019-transparency-market-research-228593931.html)

- Pujols, Y., Seal, B. N., & Meston, C. M. (2010). The association between sexual satisfaction and body image in women. *Journal of Sexual Medicine*, 7, 905–916.
- Reissing, E. D., Binik, Y. M., & Khalife, S. (1999). Does vaginismus exist? A critical review of the literature. *Journal of Nervous and Mental Disease*, 187, 261–274.
- Renshaw, D. C. (2001). Women coping with a partner's sexual avoidance. *Family Journal—Counseling & Therapy for Couples and Families*, 9, 11–16.
- Richters, J., Altman, D., Badcock, P. B., Smith, A. M. A., de Visser, R. O., Grulich, A. E., ... Simpson, J. M. (2014). Sexual identity, sexual attraction and sexual experience: the Second Australian Study of Health and Relationships. *Sexual Health*, 11, 451–460.
- Richters, J., de Visser, R. O., Badcock, P. B., Smith, A. M., Rissel, C., Simpson, J. M., & Grulich, A. E. (2014). Masturbation, paying for sex and other sexual activities: The Second Australian Study of Health and Relationships. *Sexual Health*, 11(5), 461–471. doi:10.1071/SH14116
- Richters, J., de Visser, R. O., Rissel, C. E., Grulich, A. E., & Smith, A. M. (2008). Demographic and psychosocial features of participants in bondage and discipline, "sodomasochism" or dominance and submission (BDSM): Data from a national survey. *Journal of Sexual Medicine*, 7, 1660–1668.
- Rosen, R. C., Leiblum, S. R., & Spector, I. (1994). Psychologically based treatment for male erectile disorder: A cognitive-interpersonal model. *Journal of Sex and Marital Therapy*, 20, 67–85.
- Rosen, R. C., Miner, M. M., & Wincze, J. P. (2014). Erectile dysfunction: Integration of medical and psychological approaches. In Y. M. Binik & K. S. K. Hall (Eds.), *Principles and Practice of Sex Therapy* (5 ed., pp. 61–88). New York: Guilford Press.
- Roughgarden, J. (2004). *Evolution's rainbow: Diversity, gender and sexuality in nature and people*. Oakland, CA: University of California Press.
- Rowland, D. L., Cooper, S. E., & Slob, A. K. (1996). Genital and psychoaffective responses to erotic stimulation in sexually functional and dysfunctional men. *Journal of Abnormal Psychology*, 105, 194–203.
- Salter, D., McMillan, D., Richards, M., Talbot, T., Hodges, J., Bentovim, A., et al. (2003). Development of sexually abusive behaviour in sexually victimised males: A longitudinal study. *The Lancet*, 361, 471–476.
- Sanchez, D. T., & Kiefer, A. K. (2007). Body concerns in and out of the bedroom: Implications for sexual pleasure and problems. *Archives of Sexual Behavior*, 36, 808–820.
- Sbrocco, T., Weisberg, R. B., Barlow, D. H., & Carter, M. M. (1997). The conceptual relationship between panic disorder and male erectile dysfunction. *Journal of Sex and Marital Therapy*, 23, 212–220.
- Schmitt, D. P., Alcalay, L., Allik, J., Ault, L., Austers, I., Bennett, K. L., et al. (2003). Universal sex differences in the desire for sexual variety: Tests from 52 nations, 6 continents and 13 islands. *Journal of Personality and Social Psychology*, 85, 85–104.
- Segraves, R. T. (2003). Recognizing and reversing sexual side effects of medications. In S. B. Levine, C. B. Candace, et al. (Eds.), *Handbook of clinical sexuality for mental health professionals* (pp. 377–391). New York: Brunner-Routledge.
- Segraves, R. T. (2010). Considerations for an evidence-based definition of premature ejaculation in the DSM-V. *Journal of Sexual Medicine*, 7, 672–679.
- Segraves, R. T., & Althof, S. (1998). Psychotherapy and pharmacotherapy of sexual dysfunctions. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work*. New York: Oxford University Press.
- Seto, M. C. (2009). Pedophilia. *Annual Review of Clinical Psychology*, 5, 391–407.
- Shifren, J. L., Monz, B. U., Russo, P. A., Segreti, A., & Johannes, C. B. (2008). Sexual problems and distress in United States women: Prevalence and correlates. *Obstetrics and Gynecology*, 112, 970–978.
- Spengler, A. (1977). Manifest sadomasochism of males: Results of an empirical study. *Archives of Sexual Behavior*, 6, 441–456.
- Spitzer, R. L., Gibbon, M., Skodol, A. E., Williams, J. B. W., & First, M. B. (Eds.). (1994). *DSM-IV casebook: A learning companion to the Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Press.
- Stein, M. B., Yehuda, R., Koverola, C., & Hanna, C. (1997). Enhanced dexamethasone suppression of plasma cortisol in adult women traumatized by childhood sexual abuse. *Biological Psychiatry*, 42, 680–686.
- Stevenson, J., & Jones, I. H. (1972). Behavior therapy technique for exhibitionism: A preliminary report. *Archives of General Psychiatry*, 27, 839–841.
- Suchy, Y., Eastvold, A. D., Strassberg, D. S., & Franchow, E. I. (2014). Understanding processing speed weaknesses among pedophilic child molesters: Response style vs. neuropathology. *Journal of Abnormal Psychology*, 123, 273–285.
- ter Kuile, M. M., & Reissing, E. D. (2014). Lifelong vaginismus. In Y. M. Binik & K. S. K. Hall (Eds.), *Principles and practice of sex therapy* (5th ed., pp. 177–194). New York: Guilford Press.
- ter Kuile, M. M., Both, S., & van Lankveld, J. J. (2012). Sexual dysfunctions in women. In P. Sturmey & M. Hersen (Eds.), *Handbook of evidence-based practice in clinical psychology, adult disorders* (Vol. 2, pp. 413–436). Hoboken, NJ: John Wiley & Sons.
- Thibaut, F., De La Barra, F., Gordon, H., Cosyns, P., & Bradford, J. M. (2010). The World Federation of Societies of Biological Psychiatry (WFSBP) guidelines for the biological treatment of paraphilias. *World Journal of Biological Psychiatry*, 11, 604–655.
- Tiefer, L., Hall, M., & Tavis, C. (2002). Beyond dysfunction: A new view of women's sexual problems. *Journal of Sex and Marital Therapy*, 28, 225–232.
- van Lankveld, J. J., Granot, M., Weijmar Schultz, W. C., Binik, Y. M., Wesselmann, U., Pukall, C. F., et al. (2010). Women's sexual pain disorders. *Journal of Sexual Medicine*, 7, 615–631.

- Vance, S., Cohen-Kettenis, P., Drescher, J., Meyer-Bahlburg, H., Pfafflin, F., & Zucker, K. (2010). Opinions about the DSM gender identity disorder diagnosis: Results from an international survey administered to organizations concerned with the welfare of transgender people. *International Journal of Transgenderism*, 12, 1–14.
- Vanwesenbeeck, I., Bakker, F., & Gesell, S. (2010). Sexual health in the Netherlands: Main results of a population survey among Dutch adults. *International Journal of Sexual Health*, 22, 55–71.
- von Krafft-Ebing, R. (1902). *Psychopathia sexualis*. Brooklyn, NY: Physicians and Surgeons Books.
- Wakefield, J. C. (2011). Should uncomplicated bereavement-related depression be reclassified as a disorder in the DSM-5? Response to Kenneth S. Kendler's statement defending the proposal to eliminate the bereavement exclusion. *Journal of Nervous and Mental Disease*, 199, 203–208.
- Waldinger, M. D., Quinn, P., Dilleen, M., Mundayat, R., Schweitzer, D. H., & Boolell, M. (2005). A multinational population survey of intravaginal ejaculation latency time. *Journal of Sexual Medicine*, 2, 492–497.
- Ward, T. B., & Beech, A. (2006). An integrated theory of sexual offending. *Aggression and Violent Behavior*, 11, 44–63.
- Weisberg, R. B., Brown, T. A., Wincze, J. P., & Barlow, D. H. (2001). Causal attributions and male sexual arousal: The impact of attributions for a bogus erectile difficulty on sexual arousal, cognitions, and affect. *Journal of Abnormal Psychology*, 110, 324–334.
- Wille, R., & Boulanger, H. (1984). 10 years' castration law in Schleswig-Holstein. *Beitrage zur gerichtlichen Medizin*, 42, 9–16.
- Wincze, J. P., & Barlow, D. H. (1997). *Enhancing sexuality: A problem-solving approach*. Boulder, CO: Graywind Publications.
- Wolfe, V. V. (1990). Sexual abuse of children. In A. S. Bellack, M. Hersen & A. E. Kazdin (Eds.), *International handbook of behavior modification and therapy* (2nd ed., pp. 707–729). New York: Plenum.
- Wolpe, J. (1958). *Psychotherapy by reciprocal inhibition*. Stanford, CA: Stanford University Press.
- Woo, J. S., Brotto, L. A., & Gorzalka, B. B. (2011). The role of sex guilt in the relationship between culture and women's sexual desire. *Archives of Sexual Behavior*, 40, 385–394.
- Wouda, J. C., Hartman, P. M., Bakker, R. M., Bakker, J. O., van de Wiel, H. B. M., & Schultz, W. C. (1998). Vaginal plethysmography in women with dyspareunia. *Journal of Sex Research*, 35, 141–147.
- Wylie, K. R. (1997). Treatment outcome of brief couple therapy in psychogenic male erectile disorder. *Archives of Sexual Behavior*, 26, 527–545.
- Wylie, K. R., & MacInnes, I. (2005). Erectile dysfunction. In R. Balon & R. T. Segraves (Eds.), *Handbook of sexual dysfunctions and paraphilias*. Boca Raton, FL: Taylor and Francis Group.
- Zimmer, D. (1987). Does marital therapy enhance the effectiveness of treatment for sexual dysfunction? *Journal of Sex and Marital Therapy*, 13, 193–209.

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 Table 9.5: © Professor Bernadette McSherry

# Disorders of childhood

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 10.1** describe the issues in the diagnosis of psychopathology in children
  - 10.2** discuss the description, aetiology and treatments for externalising disorders, including ADHD and conduct disorder
  - 10.3** discuss the description, aetiology and treatments for internalising disorders, including depression and anxiety disorders
  - 10.4** understand the learning disorders dyslexia and dyscalculia, and learn the causes and treatments for dyslexia
  - 10.5** discuss the description and diagnosis of intellectual disability and the current research on causes and treatments
  - 10.6** describe the symptoms, causes and treatments for autism spectrum disorders.
-

## OPENING SCENARIO

'Eric. Eric? Eric!' His teacher's voice and the laughter of his classmates roused the boy from his reverie. Glancing at the book of the girl sitting next to him, he noticed that the class was pages ahead of him. He was supposed to be answering a question about the Gold Fever at Sovereign Hills, but he had been lost in thought, wondering about what seats he and his father would have for the footy game they'd be attending that evening. A tall, lanky 12-year-old, Eric had just begun Year 7. His history teacher had already warned him about being late to class and not paying attention, but Eric just couldn't seem to get from one class to the next without stopping for drinks of water or to investigate an altercation between classmates. In class, he was rarely prepared to answer when the teacher called on him, and he usually forgot to write down the homework assignment. He already had a reputation among his peers as an 'airhead'.

Eric's relief at the sound of the bell was quickly replaced by anxiety as he reached the playground for physical education. Despite his speed and physical strength, Eric was always picked last for sport teams. His cricket team was up to bat first, and Eric sat down to wait his turn. Absorbed in studying a pile of pebbles at his feet, he failed to notice it was time to come off the pitch. The other team had already come out on field before Eric had noticed — too late to avoid the irate yells of his PE teacher. It was yet another embarrassing day at school.

At home, Eric's father told him he had to finish his homework before they could go to the Bulldogs game. He had only one page of maths problems and was determined to finish them quickly. Thirty minutes later, his father emerged from the shower to find Eric building an elaborate Lego structure on the floor of his room; the maths homework was only half done. In exasperation, Eric's father left for the game without him.

At bedtime, frustrated and discouraged, Eric was unable to sleep. He often lay awake for what seemed like hours, reviewing the disappointments of the day and berating himself for his failures. On this night, he ruminated about his lack of friends, the frustration of his teachers, and his parents' exhortations to pay attention and 'get it together'. Feeling hopeless about doing better, despite his daily resolve, Eric often found his thoughts turning to suicide. Tonight he reviewed his fantasy of wandering out into the street in front of a passing car. Although Eric had never acted on his suicidal thoughts, he frequently replayed in his mind his parents' sorrow and remorse, his classmates' irritation with him, and the concern of his teachers.

### QUESTIONS

1. If he could, how might Eric explain what he is experiencing to his father?
2. Describe what Eric's father might be thinking in this scenario.

## Introduction

Childhood disorders, like adult disorders, involve a combination of behavioural, cognitive, genetic, neurobiological and social factors in their aetiology and treatment. The number of children diagnosed with and treated for different psychological disorders has dramatically increased in recent years, but not without controversy (see focus on discovery 10.2 later in the chapter). For example, the number of people receiving a diagnosis of ADHD (attention-deficit hyperactivity disorder) in Australia increased from 7.8 percent of children in 2003 to 11 percent by 2011 (Duff, n.d.)!

In this chapter we discuss several of the disorders that are most likely to arise in childhood and adolescence. We first consider disorders involving inattention, impulsivity and disruptive behaviour, followed by depression and anxiety disorders. Finally, we discuss disorders in which the acquisition of cognitive, language, motor or social skills is disturbed. These include learning disorders as well as the most severe of developmental disorders, intellectual disability and autism spectrum disorder, which are usually chronic, persisting into adulthood.

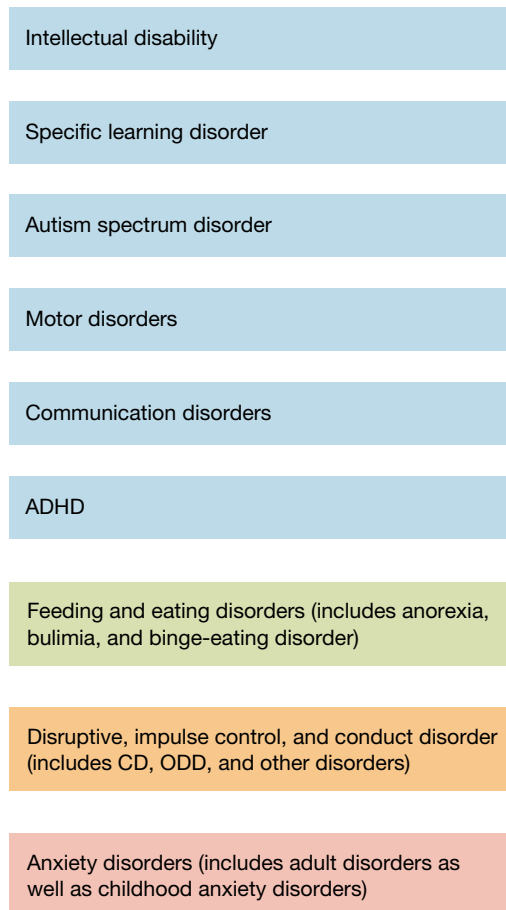
## 10.1 Classification and diagnosis of childhood disorders

**LEARNING OUTCOME 10.1** Describe the issues in the diagnosis of psychopathology in children.

Before making a diagnosis of a particular disorder in children, clinicians must first consider what is typical for a particular age. The children who lie on the floor kicking and screaming when they don't get their way would be assessed differently at age two than at age seven. The field of **developmental psychopathology** focuses on the disorders of childhood within the context of life-span development, enabling us to identify behaviours that are considered appropriate at one stage but not at another.

The childhood disorders are shown in figure 10.1. In DSM-5, most of the childhood disorders are presented in two chapters: 'Neurodevelopmental disorders' and 'Disruptive, impulse-control, and conduct disorders'. Eating disorders (which we cover separately in another chapter) are in their own chapter in DSM-5. Other disorders, such as separation anxiety disorder, are in the DSM-5 chapter on anxiety disorders.

**FIGURE 10.1** DSM-5 childhood disorders



The more prevalent childhood disorders are often divided into two broad domains: externalising disorders and internalising disorders. **Externalising disorders** are characterised by more outward-directed behaviours, such as aggressiveness, non-compliance, overactivity and impulsiveness; the category includes

attention-deficit hyperactivity disorder, conduct disorder and oppositional defiant disorder. **Internalising disorders** are characterised by more inward-focused experiences and behaviours, such as depression, social withdrawal and anxiety; the category includes childhood anxiety and mood disorders. Children and adolescents may exhibit symptoms from both domains, as described in the opening scenario about Eric.

The behaviours that comprise externalising and internalising disorders are prevalent across many countries and cultures (Polanczyk, de Lima, Horta, Biederman, & Rohde, 2007), such as Switzerland (Steinhausen & Metzke, 1998), Australia (Achenbach, Hensley, Phares, & Grayson, 1990), Puerto Rico (Achenbach et al., 1990), Kenya (Weisz, Sigman, Weiss, & Mosk, 1993), and Greece (MacDonald, Tsiantis, Achenbach, Motti-Stefanidi, & Richardson, 1995). Focus on discovery 10.1 discusses the possible role of culture in the prevalence of these problem behaviours in children.

#### FOCUS ON DISCOVERY 10.1

##### The role of culture in internalising and externalising behaviour problems

The values of a culture may play a role in whether a certain pattern of child behaviour develops or is considered a problem. One study found that in Thailand, children with internalising behaviour problems, such as fearfulness, were the ones most likely to be seen in clinics, whereas in Western countries, those with externalising behaviour problems, such as aggressiveness and hyperactivity, were more commonly seen (Weisz, Suwanlert, Wanchai, & Bernadette, 1987). The researchers attributed these differences to the fact that Buddhism, which disapproves of and discourages aggression, is widely practised in Thailand. In other words, cultural sanctions against acting out in aggressive ways may have kept these behaviours from developing at the rate that they do in Western countries like Australia, New Zealand, Canada and the United States. One of the issues in this study was that the researchers only used assessment measures that were normed on local samples, leaving open the possibility that behaviour differences between the two cultures were missed because they were not validly assessed for both cultures (see the chapter on diagnosis and assessment for more discussion of culture and assessment).

Young Thai novice monks receiving instruction. Buddhist culture may contribute to the relatively low prevalence of externalising disorders in Thailand.



Indeed, findings from a follow-up study suggest that the behaviour problems described in the same terms may not really be exactly the same across Eastern and Western cultures (Weisz, Weiss, Suwanlert, & Wanchai, 2003). The researchers compared specific behaviour problems (e.g., somatic complaints, aggressive behaviour) and broad domains (internalising, externalising) using Western and Eastern assessment measures. The broad domains of internalising and externalising behaviours were found to be the same in the Eastern and Western children, but more specific categories within those domains were not. Among boys, somatic complaints were seen consistently across cultures, but shyness was seen less consistently. Among girls, shyness was seen consistently across cultures but verbal aggressive behaviour was not.

These studies point to the importance of studying psychopathology across cultures. It is dangerous to assume that the measures we develop to assess psychopathology in Australia or New Zealand will work equally well across cultures. As the investigators cited above point out, our theories about the causes of psychopathology need to be able to take into account cultural variation in such factors as parenting practices, beliefs and values, and the ways in which parents report on their child's behaviour problems. This remains an urgent and important challenge for our field.

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### QUESTIONS

1. Why do internalising behaviour patterns differ across countries?
2. Why might it be dangerous to assume that measures developed to assess psychopathology in New Zealand will certainly work in another country, such as India?

## 10.2 Externalising disorders

**LEARNING OUTCOME 10.2** Discuss the description, aetiology and treatments for externalising disorders, including ADHD and conduct disorder.

### Attention-deficit hyperactivity disorder

The term *hyperactive* is familiar to most people, especially parents and teachers. The child who is constantly in motion — tapping fingers, jigglng legs, poking others for no apparent reason, talking out of turn and fidgeting — is often called hyperactive. Often, these children also have difficulty concentrating on the task at hand for an appropriate period of time. When such problems are severe and persistent enough, these children may meet the criteria for diagnosis of **attention-deficit hyperactivity disorder (ADHD)**.

#### Clinical descriptions of ADHD

What distinguishes the typical range of hyperactive behaviours from a diagnosable disorder? When these behaviours are extreme for a particular developmental period, persistent across different situations, and linked to significant impairments in functioning, the diagnosis of ADHD may be appropriate (Hinshaw & Scheffler, 2014). The DSM-5 criteria are presented below.

Children with ADHD seem to have particular difficulty controlling their activity in situations that call for sitting still, such as in the classroom or at mealtimes. When asked to be quiet, they appear unable to stop moving or talking. Their activities and movements may seem haphazard. They may quickly wear out their shoes and clothing, smash their toys, and exhaust their families and teachers.

Many children with ADHD have inordinate difficulty getting along with peers and establishing friendships (Blachman & Hinshaw, 2002; Hinshaw & Melnick, 1995), perhaps because their behaviour is often aggressive and intrusive. Although these children are usually friendly and talkative, they often miss subtle social cues, such as noticing when other children are tiring of their constant jigglng. Unfortunately, children with ADHD often overestimate their ability to navigate social

situations with peers (Hoza, Murray-Close, Arnold et al., 2010). A longitudinal study of children with and without ADHD who were followed up every year for six years found that poor social skills, aggressive behaviour, and self-overestimation of performance in social situations all predicted problems with peers up to six years later. The researchers also found what they called ‘vicious cycles’ with these three domains — poor social skills, aggressive behaviour and overestimation of one’s social abilities — all in turn predicted a decline in these abilities at the next follow-up, which in turn predicted greater problems with peers at the next follow-up (Murray-Close, Hoza, Hinshaw et al., 2010).

In another study, children were asked to instant-message (IM) other children in what appeared to be an online chat room (Mikami, Huang-Pollack, Pfiffner, McBurnett, & Hangai, 2007). Actually, children were interacting with four simulated peers on the computer, and thus all children got the same IMs from the simulated peers. The researchers coded the messages and the participants’ reported experiences of the chat elicited in subsequent interviews. Children with ADHD were more likely to IM statements that were hostile and off the topic than were children without ADHD, and children’s chat room experiences were related to other measures of social skills difficulties, suggesting that this common way of ‘interacting’ with peers, even though not face-to-face, is also impaired among children with ADHD.

## DSM-5

### DSM-5 criteria for attention-deficit hyperactivity disorder

- Either (a) or (b):
  - (a) the individual experiences six or more manifestations of inattention present for at least six months to a maladaptive degree and greater than what would be expected given a person’s developmental level; for example, careless mistakes, not listening well, not following instructions, easily distracted, forgetful in daily activities
  - (b) the individual experiences six or more manifestations of hyperactivity-impulsivity present for at least six months to a maladaptive degree and greater than what would be expected given a person’s developmental level; for example, fidgeting, running about inappropriately (in adults, restlessness), acting as if ‘driven by a motor’, interrupting or intruding, incessant talking.
- Several of the above symptoms present before age 12.
- Symptoms are present in two or more settings, for example, at home, school or, work.
- Individuals experience significant impairment in social, academic or occupational functioning.
- For people age 17 or older, only five signs of inattention and/or five signs of hyperactivity-impulsivity are needed to meet the diagnosis.

Children with ADHD are often singled out very quickly and rejected or neglected by their peers. For example, in a study of previously unacquainted boys at a summer camp, boys with ADHD who exhibited a number of externalising behaviours, such as overt aggression and non-compliance, were regarded quite negatively by their peers during the first day of camp, and these impressions remained unchanged throughout the six-week camp period (Erhardt & Hinshaw, 1994; Hinshaw, Zupan, Simmel, Nigg, & Melnick, 1997).

### ADHD in the DSM-5 and comorbidities

Two changes in the DSM-5 criteria for ADHD may result in more children and adults receiving the diagnosis. First, the age of onset was changed from under age 7 to under age 12. This means that children who begin to show symptoms after age seven can now meet the diagnosis for ADHD. Second, adults only need to show symptoms in five domains instead of the six required for children.

The DSM-5 includes three specifiers to indicate which symptoms predominate:

1. *predominantly inattentive*: children whose problems are primarily those of poor attention
2. *predominantly hyperactive-impulsive*: children whose difficulties result primarily from hyperactive/impulsive behaviour
3. *combined*: children who have both sets of problems.

The combined specifier comprises the majority of children with ADHD. These children are more likely than those with other subtypes to develop conduct problems and oppositional behaviour, to be placed in special classes for children with behaviour problems, and to have difficulties interacting with their peers (Faraone, Biederman, Weber, & Russell, 1998). Children with the predominantly inattentive specifier have more difficulties with focused attention or speed of information processing (Barkley, Grodzinsky, & DuPaul, 1992), perhaps associated with problems involving dopamine and the prefrontal cortex (Volkow et al., 2009).

ADHD and conduct disorder (discussed later in the chapter) frequently co-occur and share some features in common (Beauchaine, Hinshaw, & Pang, 2010). There are some differences, however. ADHD is associated more with off-task behaviour in school, cognitive and achievement deficits, and a better long-term prognosis.

When these two disorders occur in the same child, the worst features of each are manifest. Such children exhibit the most serious antisocial behaviour, are most likely to be rejected by their peers, have the worst academic achievement, and have the poorest prognosis (Hinshaw & Lee, 2003). Girls with both ADHD and conduct disorder exhibit more antisocial behaviour, other psychopathology, and risky sexual behaviour than girls with only ADHD (Monuteaux, Faraone, Gross, & Biederman, 2007).

Internalising disorders, such as anxiety and depression, also frequently co-occur with ADHD. Estimates suggest that as many as 30 percent of children with ADHD may have comorbid internalising disorders (e.g., Jensen, Martin, D., & Cantwell, 1997; MTA Cooperative Group, 1999b). In addition, about 15 to 30 percent of children with ADHD have a learning disorder (Barkley, DuPaul, & McMurray, 1990; Casey, Rourke, & Del Dotto, 1996).

Although having both ADHD and conduct disorder is associated with substance use, a prospective study found that the hyperactive symptoms of ADHD predicted subsequent substance use (nicotine, alcohol, illicit drugs) at age 14 and substance use disorder at age 18 even after controlling for symptoms of conduct disorder, and this was equally true for boys and girls (Elkins, McGue, & Iacono, 2007).

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Stephen Hinshaw, a renowned developmental psychopathology researcher and expert on mental illness stigma, is conducting one of the largest ongoing studies of girls with ADHD.



## Prevalence

By some accounts, the prevalence of ADHD has risen dramatically in the past decade, ranging from 8 to 11 percent compared to older estimates of 3 to 7 percent (Merikangas, et al., 2010). As well, there are a variety of prevalence rates given by different studies. For example, in Australia, some studies have listed a rate of 2.4 percent and others as much as 11 percent (Duff, n.d.; Faraone, Sergeant, Gillberg, & Biederman, 2003). Why the difference in rates? Several explanations are possible, suggesting that the increase is due to factors other than an actual increase in the disorder. For example, most children receive a diagnosis after a brief visit with a pediatrician, but correct diagnoses require careful and thorough assessments (Hinshaw & Scheffler, 2014). Thus, many children may be getting the diagnosis when it is not warranted.

Secondly, some countries report much higher rates than others. In Sweden, it has been reported that the prevalence of ADHD in children is as low as 3.7 percent but in countries such as Germany or Colombia, the reported rate can be as high as 17.8 percent (Faraone et al., 2003)!

### Sex differences

Despite the controversy over the increasing prevalence rates of ADHD, much evidence indicates that ADHD is three times more common in boys than in girls (Merikangas et al., 2010). Until recently, very few carefully controlled studies of girls with ADHD were conducted. Two groups of researchers have conducted large, careful studies of ADHD in girls (Biederman & Faraone, 2004; Hinshaw, 2002). Here are some of the key findings at the initial assessment and then again five years later (Hinshaw, Carte, Sami, Treuting, & Zupan, 2002; Hinshaw, Owens, Sami & Fargeon, 2006) and 10 to 11 years later (Biederman et al., 2010; Hinshaw et al., 2012).

- Girls with the DSM-IV-TR combined subtype (now a specifier in DSM-5) were more likely to have a comorbid diagnosis of conduct disorder or oppositional defiant disorder than girls without ADHD, and this difference remained five years after initial diagnosis.
- Girls with ADHD were viewed more negatively by peers than girls without ADHD.
- Girls with ADHD were likely to be more anxious and depressed than were girls without ADHD, and this remained true five years after initial diagnosis and into early adulthood.
- Girls with ADHD exhibited a number of neuropsychological deficits, particularly in executive functioning (e.g., planning, solving problems), compared with girls without ADHD.
- By adolescence, girls with ADHD were more likely to have symptoms of an eating disorder and substance abuse than girls without ADHD (Mikami, Hinshaw, Arnold et al., 2010), but not by early adulthood.

### ADHD in adulthood

At one time it was thought that ADHD simply went away by adolescence. However, this belief has been challenged by numerous longitudinal studies (Barkley, Fischer, Smallish, & Fletcher, 2002; Hinshaw et al., 2006; Lee, Lahey, Owens, & Hinshaw, 2008; Weiss & Hechtman, 1993). Although some children show reduced severity of symptoms in adolescence and early adulthood, 65 to 80 percent of children with ADHD still have symptoms associated with impairments in adolescence (Biederman et al., 2006; Hart, Lahey, Loeber, Applegate, & Frick, 1995; Hinshaw et al., 2006). Table 10.1 provides a catalogue of behaviours that are found more often among adolescents with ADHD than among adolescents without it. Many children with ADHD do not

Aggression is common among boys with ADHD, and it contributes to their being rejected by peers.



appear to take a ‘hit’ with respect to academic achievement, however — many studies indicate that achievement is within the average range for both adolescent boys (Lee et al., 2008) and girls (Hinshaw et al., 2006).

**TABLE 10.1 Behaviours in adolescents with and without ADHD**

Behaviour	Percentage of adolescents who show this behaviour	
	With ADHD	Without ADHD
Blurts out answers	65.0	10.6
Is easily distracted	82.1	15.2
Doesn't complete tasks before moving to another	77.2	16.7
Doesn't sustain attention	79.7	16.7
Doesn't follow instructions	83.7	12.1
Doesn't listen to others well	80.5	15.2
Engages in physically dangerous activities	37.4	3.0
Fidgets	73.2	10.6
Finds it hard to play quietly	39.8	7.6
Gets out of seat often	60.2	3.0
Interrupts others	65.9	10.6
Loses things needed for tasks	62.6	12.1
Talks a lot	43.9	6.1

*Source:* Adapted from Barkley, Du Paul, & McMurray (1990).

In adulthood, most people with ADHD are employed and financially independent, but some studies have found that adults with ADHD are generally at a lower socioeconomic level and change jobs more frequently than is typical (Mannuzza, Klein, Bonagura, Malloy, Giampino, & Addalli, 1991; Weiss & Hechtman, 1993). Findings from a review of the studies that have assessed ADHD longitudinally into adulthood indicate that up to 15 percent of people continued to meet DSM criteria as 25-year-old adults. Even more people — close to 60 percent — continue to exhibit symptoms that are associated with impairment in several domains (Faraone, Biederman, & Mick, 2005; Hinshaw et al., 2012). Thus, ADHD symptoms may decline with age, but they do not entirely go away for many people.

## Aetiology of ADHD

### Genetic factors

Substantial evidence indicates that genetic factors play a role in ADHD (Thapar, Langley, Owen, & O'Donovan, 2007). Adoption studies (e.g., Sprich, Biederman, Crawford, Mundy, & Faraone, 2000) and numerous large-scale twin studies (e.g., Levy, Hay, McStephen, Wood, & Waldman, 1997; Sherman, Iacono, & McGue, 1997) indicate that ADHD has a genetic component, with heritability estimates as high as 70 to 80 percent (Sullivan, Daly, & O'Donovan, 2012). Molecular genetics studies that seek to discover the multiple genes linked to ADHD have revealed a number of candidate genes. Most notable are those genes associated with the neurotransmitter dopamine. Specifically, three different dopamine genes have been implicated in ADHD and have modest effect sizes in meta-analyses: dopamine receptor genes called *DRD4* and *DRD5*, and a dopamine transporter gene called *DAT1* (Gizer, Ficks, & Waldman, 2009). An additional gene, *SNAP-25*, which codes for a protein that promotes plasticity (i.e., adaptability) of neuron synapses, has also been associated with ADHD (Forero, Arboleda, Vasquez, & Arboleda 2009; Gizer et al., 2009).

Even with these promising findings, most investigators agree that a single gene will not ultimately be found to account for ADHD (Nigg, 2013). Rather, several genes interacting with each other and with environmental factors will provide the most complete picture of the role of genes in ADHD. For example, studies have found that the *DRD4* or *DAT1* genes are associated with increased risk of ADHD only among those who also had particular environmental factors — namely, prenatal maternal nicotine or alcohol use (Brookes et al., 2006; Neuman et al., 2007). In addition, polygene (i.e., multiple genes; see chapter 1) scores derived from meta-analyses of genetic factors are higher in those with ADHD compared to those without ADHD (Hamshere et al., 2013).

### Neurobiological factors

Studies suggest that brain structure, function and connectivity differ in children with and without ADHD, particularly in areas of the brain linked to the neurotransmitter dopamine. For example, studies of brain structure have found that dopaminergic areas of the brain, such as the caudate nucleus, globus pallidus and frontal lobes, are smaller in children with ADHD than in children without ADHD (Castellanos et al., 2002; Swanson, Kinsbourne, Nigg et al., 2007). A meta-analysis of 55 brain-imaging studies found that children with ADHD exhibit less activation in frontal areas of the brain (Cortese et al., 2012). Moreover, children with ADHD perform poorly on neuropsychological tests that rely on the frontal lobes (such as inhibiting behavioural responses), providing further support for the theory that a basic deficit in this part of the brain may be related to the disorder (Nigg & Casey, 2005).

Other neurobiological risk factors for ADHD include a number of perinatal and prenatal complications. Low birth weight, for example, is a predictor of the development of ADHD (e.g., Bhutta, Cleves, Casey, Cradock, & Anand, 2002; Breslau et al., 1996; Whitaker et al., 1997). However, the impact of low birth weight on later symptoms of ADHD can be mitigated by greater maternal warmth (Tully, Arseneault, Caspi, Moffitt, & Morgan, 2004).

### Environmental toxins

Early theories of ADHD in the 1970s involved the role of environmental toxins. Feingold (1973) proposed that additives and artificial colours in foods upset the central nervous systems of children who were hyperactive, and he prescribed a diet free of them. However, well-controlled studies of the so-called Feingold diet have found that very few children with ADHD respond positively to it. More recent and better-designed studies have found that elements of the diet, particularly additives, may influence ADHD symptoms for a subset of children. Two meta-analyses reported small effect sizes for artificial food colouring on hyperactive behaviour among children with ADHD (Nigg, Lewis, Edinger, & Falk, 2012; Schnab & Trinh, 2004). Thus, there is limited evidence that food additives impact hyperactive behaviour. The popular view that refined sugar can cause ADHD has not been supported by careful research (Wolraich, Wilson, & White, 1995).

Lead is another environmental toxin that has been studied. Some evidence suggests that higher blood levels of lead may be associated to a small degree with symptoms of hyperactivity and attentional problems (Braun, Kahn, Froelich, Auinger, & Lanphear, 2006), as well as with the diagnosis of ADHD. However, most children with higher blood levels of lead do not develop ADHD, and most children with ADHD do not show such elevated blood levels. Nevertheless, given the unfortunate frequency with

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Michael Phelps, who won 23 Olympic gold medals in swimming, also struggled with ADHD as a child.



which children are exposed to low levels of lead, investigators continue to examine how lead exposure might play a role, perhaps by influencing other cognitive abilities.

Nicotine — specifically, maternal smoking — is an environmental toxin that may play a role in the development of ADHD. A review of 24 studies examining the association between maternal smoking and ADHD found that exposure to tobacco in utero was associated with ADHD symptoms (Linnet, Dalsgaard, Obel et al., 2003). However, an interesting study calls this linkage into question. Thapar and colleagues (2009) examined ADHD symptoms in the offspring of two groups of mothers who were regular smokers when pregnant. One group of smoking mothers delivered babies that were not genetically related to them (e.g., surrogate mother delivering a genetically unrelated baby for another family); the other group of mothers delivered babies that were genetically related. The researchers reasoned that if maternal smoking during pregnancy was an important factor in ADHD, smoking ought to be related to ADHD symptoms in the offspring from both groups of mothers. By contrast, if genetic factors were important, then the association between smoking and ADHD symptoms ought to be higher in the offspring of genetically related mothers. They found that ADHD symptoms were related to maternal smoking in both groups, but the association was significantly higher in children whose genetically related mothers smoked during pregnancy. These findings suggest that smoking might not be a causal factor by itself but that it is related to other maternal behaviour and psychopathology that might increase the risk of ADHD.

### Family factors in ADHD

Family factors are also important in ADHD, particularly in their interaction with neurobiological factors. For example, the parent–child relationship interacts with neurobiological factors in a complex way to contribute to ADHD symptom expression (Hinshaw et al., 1997). Just as parents of children with ADHD may give them more commands and have negative interactions with them (Anderson, Hinshaw, & Simmel, 1994; Heller, Baker, Henker, & Hinshaw, 1996), so these children have been found to be less compliant and more negative in interactions with their parents (Barkley, Karlsson, & Pollard, 1985; Tallmadge & Barkley, 1983). Certainly, it must be difficult to parent a child who is impulsive, aggressive, non-compliant and unable to follow instructions. As we will discuss shortly, when children with ADHD take stimulant medication, either alone or in combination with behavioural treatment, parents' commands, negative behaviour and ineffective parenting decrease (Barkley, 1990; Wells, Epstein, Hinshaw et al., 2000), suggesting that the child's behaviour has at least some negative effect on the parents' behaviour.

It is also important to consider a parent's own history of ADHD. As noted earlier, ADHD appears to have a substantial genetic component. Thus, it is not surprising that many parents of children with ADHD have ADHD themselves. In one study that examined couples' parenting practices with their ADHD children, fathers who had a diagnosis of ADHD were less effective parents, suggesting that parental psychopathology may make parenting all the more difficult (Arnold, O'Leary, & Edwards, 1997). Family characteristics thus may well contribute to maintaining or exacerbating the symptoms and consequences of ADHD; however, there is little evidence to suggest that families actually cause ADHD (Johnston & Marsh, 2001).

### Treatment of ADHD

We now turn to treatments. ADHD is typically treated with medication and with behavioural therapies based on operant conditioning.

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Children born to mothers who smoked cigarettes during pregnancy have an increased risk of ADHD.



### **Stimulant medications**

Stimulant medications, such as methylphenidate, known as Ritalin or Attenta, have been prescribed in Australia and New Zealand for ADHD for more than 50 years. A recent report from a company that managed 90 million prescriptions in the United States found that more than 80 percent of children with ADHD had been given a prescription for stimulant medications, including almost 10 percent of all adolescent boys (Express Scripts Lab, 2014). The prescription of these medications often continues into adolescence and adulthood in light of the accumulating evidence that the symptoms of ADHD do not usually disappear with the passage of time. The number of adults taking stimulant medication increased more than 50 percent between 2008 and 2012 (Express Scripts Lab, 2014).

The medicines used to treat ADHD reduce disruptive behaviour and impulsivity, and improve ability to focus attention (Hinshaw & Scheffler, 2014). Numerous controlled studies comparing stimulants with placebos in double-blind designs have shown short-term improvements in concentration, goal-directed activity, classroom behaviour, and social interactions with parents, teachers and peers, as well as reductions in aggressiveness and impulsivity in about 75 percent of children with ADHD (Spencer, Biederman, Wilens, Harding, O'Donnell, & Griffin, 1996; Swanson, McBurnett, Christian & Wigal, 1995). These drugs appear to help in these domains by interacting with the dopamine system in the brain (Volkow et al., 2011).

The best designed randomised controlled trial of treatments for ADHD was the Multimodal Treatment of Children with ADHD (MTA) study. Conducted at six different sites for 14 months with nearly 600 children with ADHD, the study compared standard community-based care and three other treatments: (1) medication alone, (2) medication plus intensive behavioural treatment, involving both parents and teachers, and (3) intensive behavioural treatment alone. Across the 14-month period, children receiving medication alone had fewer ADHD symptoms than children receiving intensive behavioural treatment alone. The combined treatment was slightly superior to the medication alone and had the advantage of not requiring as high a dosage of Ritalin to reduce ADHD symptoms. In addition, the combined treatment yielded improved functioning in areas such as social skills more than did the medication alone. The medication alone and the combined treatment were superior to community-based care, though the behavioural treatment alone was not (MTA Cooperative Group, 1999a, 1999b).

Despite the initially promising findings from the MTA study, additional follow-ups from this study have not been quite as encouraging, at least where medication is concerned. Importantly, all the children maintained the gains made during the 14-month treatment, even as they all returned to receiving standard community care, and this was true at the three-, six- and eight-year follow-ups. However, children in the medication alone or the combined treatment groups were no longer doing better than children who received the intensive behavioural treatment or standard community care at the three-year follow-up (Jensen, Arnold, Swanson et al., 2007) or the six- and eight-year follow-ups (Molina et al., 2009). In other words, the relatively superior effects of medication that were observed in the combined treatment and medication alone groups did not persist beyond the study, at least for some of the children (Swanson et al., 2007).

Does this mean that medication does not work? Not necessarily. The MTA study demonstrated that carefully prescribed and managed stimulant medication is effective for children with ADHD. However, medication as it is administered in the community does not appear to offer any benefits above and beyond other forms of treatment, according to these MTA follow-up studies.

These findings are important in light of the side effects that stimulant medication can have, such as transient loss of appetite, weight loss, stomach pain and sleep problems.

### **Psychological treatment**

Other promising treatments for ADHD involve parent training and changes in classroom management (Chronis, Jones, & Raggi, 2006). These programs have demonstrated at least short-term success in improving both social and academic behaviour. In these treatments, children's behaviour is monitored at home and in school, and they are reinforced for behaving appropriately — for example, for remaining in their seats and working on assignments. Point systems and daily report cards (DRCs) are typical components of these programs. Children earn points or stars for behaving in certain ways; the children can

then spend their earnings for rewards. The DRC also allows parents to see how their child is doing in school. The focus of these programs is on improving academic work, completing household tasks, or learning specific social skills, rather than on reducing ADHD symptoms. Parent-training programs are also effective, although it is unclear whether they improve children's behaviour beyond the effects of treatment with medication (MTA Cooperative Group, 1999a, 1999b).

Findings from the MTA study indicate that intensive behavioural therapies can be very helpful to children with ADHD. In that study, some of the children participated in an intensive eight-week summer program that included a number of validated behavioural treatments. At the end of the summer program, children receiving the combined treatment had very few significant improvements over children receiving the intensive behavioural treatment alone (Arnold et al., 2003; Pelham, Gnagy, Greiner et al., 2000). This finding suggests that intensive behavioural therapy may be as effective as Ritalin combined with a less intensive behavioural therapy.

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Point systems and star charts, which are common in classrooms, are particularly useful in the treatment of ADHD.



## Conduct disorder

**Conduct disorder** is another externalising disorder. Before discussing this disorder, however, we briefly discuss two other related but less well-understood disorders.

*Intermittent explosive disorder* (IED) involves recurrent verbal or physical aggressive outbursts that are far out of proportion to the circumstances. What distinguishes IED from conduct disorder is that the aggression is impulsive and not preplanned towards other people (American Psychiatric Association, 2013). For example, a child with IED may have an aggressive outburst after not getting his or her way but would not plan aggressive retaliation.

There is some debate as to whether another DSM disorder, *oppositional defiant disorder* (ODD), is distinct from conduct disorder, a precursor to it, or an earlier and milder manifestation of it (Hinshaw & Lee, 2003; Lahey, McBurnett, & Loeber, 2000). ODD is diagnosed if a child does not meet the criteria for conduct disorder — most especially, extreme physical aggressiveness — but exhibits such behaviours as losing his or her temper, arguing with adults, repeatedly refusing to comply with requests from adults, deliberately doing things to annoy others, and being angry, spiteful, touchy or vindictive.

ODD and ADHD frequently occur together, but ODD is different from ADHD in that the defiant behaviour is not thought to arise from attentional deficits or impulsiveness. One manifestation of difference is that children with ODD are more deliberate in their unruly behaviour than children with ADHD. Although conduct disorder is three to four times more common among boys than among girls, research suggests that boys are only slightly more likely to have ODD, and some studies find no difference in prevalence rates for ODD between boys and girls (Loeber, Burke, Lahey, Winters, & Zera, 2000; Merikangas et al., 2010). Because less is known about IED and ODD, we will focus here on the more serious diagnosis of conduct disorder.

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Conduct disorder is diagnosed among those who are aggressive, steal, lie and vandalise property.



Perhaps more than any other childhood disorder, conduct disorder is defined by the impact of the child's behaviour on people and surroundings.

### **Conduct disorder in DSM-5**

The DSM-5 criteria for conduct disorder (see below) focus on aggressive behaviours (e.g., physical cruelty to people or animals), serious rule violations (e.g., truancy), property destruction and deceitfulness. Often the behaviour is marked by callousness, viciousness and lack of remorse.

DSM-5 includes a 'limited prosocial emotions' diagnostic specifier for children who have what are referred to as callous and unemotional traits. These traits refer to characteristics such as a lack of

remorse, empathy and guilt, and shallow emotions. A longitudinal study found that children with high levels of conduct problems and high levels of callous and unemotional traits had more problems with symptoms, peers, and families compared to children with conduct problems but low levels of callous and unemotional traits (Fontaine, McCrory, Boivin, Moffitt, & Viding, 2011). A comprehensive review of callous and unemotional traits in children and adolescents revealed that these traits are associated with a more severe course, more cognitive deficits, more antisocial behaviour, poorer response to treatment and perhaps distinct aetiologies (Frick, Ray, Thornton, & Kahn, 2014).

### Comorbidities and longitudinal course

Many children with conduct disorder display other problems, such as substance abuse and internalising disorders. Some research suggests that conduct disorder precedes substance use problems (Nock, Kazdin, Hiripi, & Kessler, 2006), but other findings suggest that conduct disorder and substance use occur concomitantly, with the two conditions exacerbating each other (Loeber et al., 2000).

#### DSM-5

##### DSM-5 criteria for conduct disorder

- People with conduct disorder display a repetitive and persistent behaviour pattern that violates the basic rights of others or conventional social norms as manifested by the presence of three or more of the following in the previous 12 months and at least one of them in the previous 6 months:
  - (a) aggression to people and animals, for example, bullying, initiating physical fights, physical cruelty to people or animals, forcing someone into sexual activity
  - (b) destruction of property, for example, fire-setting, vandalism
  - (c) deceitfulness or theft, for example, breaking into another's house or car, conning, shoplifting
  - (d) serious violation of rules, for example, staying out at night before age 13 in defiance of parental rules, truancy before age 13.
- Significant impairment in social, academic or occupational functioning.

Anxiety and depression are common among children with conduct disorder, with comorbidity estimates varying from 15 to 45 percent (Loeber & Keenan, 1994; Loeber et al., 2000). Evidence suggests that conduct disorder precedes depression and most anxiety disorders, with the exceptions of specific phobias and social anxiety, which appear to precede conduct disorder (Nock et al., 2006).

How early does conduct disorder begin? Studies estimate that as many as 7 percent of preschool children exhibit the symptoms of conduct disorder (Egger & Angold, 2006). One longitudinal study assessed a group of preschool children at age three and again at age six (Rolon-Arroyo, Arnold, & Harvey, 2013). Parents were interviewed using diagnostic interviews at the two time points. The researchers found that conduct disorder symptoms at age three predicted conduct disorder symptoms at age six, even when controlling for symptoms of ADHD and ODD. These findings suggest that assessing conduct disorder early is important because these symptoms are not just manifestations of typical developmentally disruptive behaviours.

Moffitt (1993) theorised that two different courses of conduct problems should be distinguished. Some people seem to show a life-course-persistent pattern of antisocial behaviour, beginning to show conduct problems by age three and continuing to commit serious transgressions into adulthood. Others are adolescence-limited — they have typical childhoods, engage in high levels of antisocial behaviour during adolescence, and have typical, non-problematic adulthoods. Moffitt proposed that the adolescence-limited form of antisocial behaviour is the result of a maturity gap between the adolescent's physical maturation and his or her opportunity to assume adult responsibilities and obtain the rewards usually accorded such behaviour.

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Children with the life-course-persistent type of conduct disorder continue to have trouble with the law into their mid-20s.



Cumulative evidence supports this distinction (Moffitt, 2007). The original sample from which Moffitt and colleagues made the life-course-persistent and adolescence-limited distinction was a sample of over 1000 people from Dunedin, New Zealand, who were assessed every 2 or 3 years from age 3 to 32. Both boys and girls with the life-course-persistent form of conduct disorder had an early onset of antisocial behaviour that persisted through adolescence and into adulthood. As children, they had a number of other problems, such as academic underachievement, neuropsychological deficits, and comorbid ADHD (Moffitt & Caspi, 2001). Other evidence supports the notion that children with the life-course-persistent type have more severe neuropsychological deficits and family psychopathology, and these findings have been replicated across cultures (Hinshaw & Lee, 2003).

Those who were classified as life-course-persistent continued to have the most severe problems, including psychopathology, poorer physical health, lower socioeconomic status, lower levels of education, partner and child abuse, and violent behaviour at age 32; this was true for men and women (Odgers et al., 2008).

Interestingly, those classified as adolescence-limited, who were expected to 'grow out' of their aggressive and antisocial behaviour, continued to have troubles with substance use, impulsivity, crime and overall mental health in their mid-20s (Moffitt, Caspi, Harrington, & Milne, 2002). Moffitt and colleagues have since suggested that *adolescent onset* is the more appropriate term for this group of people, as the conduct problems are not entirely limited to adolescence (Odgers et al., 2007). By age 32, women with the adolescent-onset type were not having difficulties with violent behaviour, but men still were. However, both men and women continued to have substance use problems, economic problems and physical health problems (Odgers et al., 2008).

### **Prevalence**

Estimates suggest that conduct disorder is fairly common, with a prevalence rate of about 2.1 percent (Lawrence et al., 2015). Like ADHD, conduct disorder is more common in boys than in girls.

### **Prognosis**

The prognosis for children diagnosed as having conduct disorder is mixed. The results just described show that men and women with the life-course-persistent type of conduct disorder will likely continue to have all sorts of problems in adulthood, including violent and antisocial behaviour. However, conduct

disorder in childhood does not inevitably lead to antisocial behaviour in adulthood. For example, another longitudinal study indicated that although about half of boys with conduct disorder did not fully meet the criteria for the diagnosis at a later assessment (one to four years later), almost all of them continued to demonstrate some conduct problems (Lahey et al., 1995).

<b>TABLE 10.2    Prevalence rates of conduct disorder in children and adolescents in Australia by sex and age group</b>			
<b>Age group</b>	<b>Males (%)</b>	<b>Females (%)</b>	<b>Persons (%)</b>
4–11 years	2.5	1.6	2.0
12–17 years	2.6	1.6	2.1
4–17 years	2.5	1.6	2.1

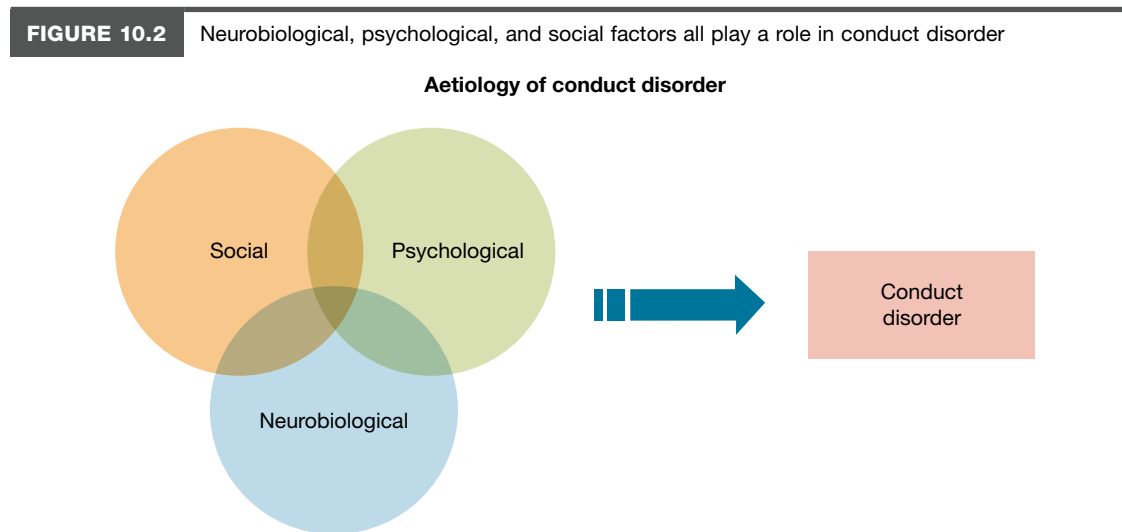
*Source:* Lawrence et al. (2015, p. 57).

## Aetiology of conduct disorder

Multiple factors are involved in the aetiology of conduct disorder, including genetic, neurobiological, psychological, and social factors that interact in a complex manner (figure 10.2). A review concluded that the evidence favours an aetiology that includes heritable temperamental characteristics that interact with other neurobiological difficulties (e.g., neuropsychological deficits) as well as with a whole host of environmental factors (e.g., parenting, school performance, peer influences) (Hinshaw & Lee, 2003).

### Genetic factors

The evidence for genetic influences in conduct disorder is mixed, although heritability likely plays a part. Part of the reason for the mixed findings is that some of the genetic influences for conduct disorder are shared with other disorders, including ADHD and depression, and some of the genetic influences are specific to conduct disorder (Lahey, Van Hulle, Singh, Waldman, & Rathouz, 2011; Lahey & Waldman, 2012).



Three large-scale adoption studies, in Sweden, Denmark and the United States, have been conducted, but two of them focused on the heritability of criminal behaviour rather than conduct disorder (Simonoff, 2001). As with most traits, these studies indicate that criminal and antisocial behaviour is accounted for by both genetic and environmental factors. Interestingly, despite different prevalence rates for boys and girls, the evidence favouring genetic and environmental contributions to conduct disorder and antisocial

behaviour does not differ between boys and girls. A meta-analysis of twin and adoption studies of anti-social behaviour indicated that 40 to 50 percent of antisocial behaviour was heritable (Rhee & Waldman, 2002).

Distinguishing types of conduct problems may help to clarify findings on the heritability of conduct disorder. Evidence from twin studies indicates that aggressive behaviour (e.g., cruelty to animals, fighting, destroying property) is more heritable than other delinquent behaviour (e.g., stealing, running away, truancy) (Burt, 2009). Other evidence suggests that the age when antisocial and aggressive behaviour problems begin is related to heritability. For example, aggressive and antisocial behaviours that begin in childhood, as in the case of Moffitt's life-course-persistent type, are more heritable than similar behaviours that begin in adolescence (Taylor, Iacono, & McGue, 2000).

One elegant study examined the interaction between genetic and environmental factors in predicting later adult antisocial behaviour (Caspi et al., 2002). It examined the *MAOA* gene, which is located on the X chromosome and releases an MAO enzyme, which metabolises a number of neurotransmitters, including dopamine, serotonin and norepinephrine. This gene varies in its activity, with some people having high and others low MAOA activity. Using a large sample of over 1000 children from Dunedin, New Zealand, the researchers measured MAOA activity and assessed the extent to which the children had been maltreated. Being maltreated as a child was not enough to predict later conduct disorder, nor was the presence of low MAOA activity. Rather, those children who were both maltreated and had low MAOA activity were more likely to develop conduct disorder than either children who were maltreated but had high MAOA activity or children who were not maltreated but had low MAOA activity. Thus, both environment and genes mattered. A meta-analysis of several such studies confirms these findings: being maltreated was linked to later antisocial behaviour only via genetics (Taylor & Kim-Cohen, 2007).

#### **Brain function, autonomic nervous system, and neuropsychological factors**

Neuroimaging studies of children with conduct disorder have revealed deficits in regions of the brain that support emotion, particularly empathetic responses. For example, children with callous and unemotional traits have difficulty perceiving distress (fear, sadness, pain) and happiness on the face of others, but do not have trouble perceiving anger (Marsh & Blair, 2008). In addition, these children show reduced activation in brain regions associated with emotion, such as the amygdala and prefrontal cortex (Blair, 2013). Children with callous and unemotional traits also do not learn to associate their behaviour with reward or punishment as easily as do other children, and this is associated with dysfunction in brain regions associated with emotion (e.g., amygdala) and reward (e.g., ventral striatum) (Blair, 2013).

Other studies indicate that autonomic nervous system abnormalities are associated with antisocial behaviour in adolescents. Specifically, lower levels of resting skin conductance and heart rate are found among adolescents with conduct disorder, suggesting that they have lower arousal levels than adolescents without conduct disorder (Ortiz & Raine, 2004; Raine, Venables, & Williams, 1990). Why does low arousal matter? Like the neuroimaging studies just discussed, these findings suggest that adolescents who exhibit antisocial behaviour may not fear punishment as much as adolescents who don't exhibit such behaviour. Thus, these children may be more likely to behave in antisocial ways without the fear that they will get caught.

Neuropsychological deficits have also been observed in children with conduct disorder (Lynam & Henry, 2001; Moffitt, Lynam, & Silvia, 1994). These deficits include poor verbal skills, difficulty with executive functioning (the ability to anticipate, plan, use self-control and solve problems) and problems with memory. In addition, children who develop conduct disorder at an earlier age (i.e., life-course-persistent type) have an IQ score of 1 standard deviation below age-matched peers without conduct disorder, and this IQ deficit is apparently not attributable to lower socioeconomic status or school failure (Lynam, Moffitt, & Stouthamer-Loeber, 1993; Moffitt & Silvia, 1988).

#### **Psychological factors**

An important part of typical child development is the growth of social emotions and moral awareness — the acquisition of a sense of what is right and wrong and the ability, even desire, to abide by rules and norms. Most people refrain from hurting others not only because it is illegal but also because it would make them feel guilty. Children with conduct disorder, particularly those with callous and unemotional

traits, seem to be deficient in this moral awareness, lacking remorse for their wrongdoing (Blair, 2013; Cimbora & McIntosh, 2003; Frick et al., 2014). In adulthood, these traits figure prominently in antisocial personality disorder and psychopathy (discussed in the chapter on personality and personality disorders).

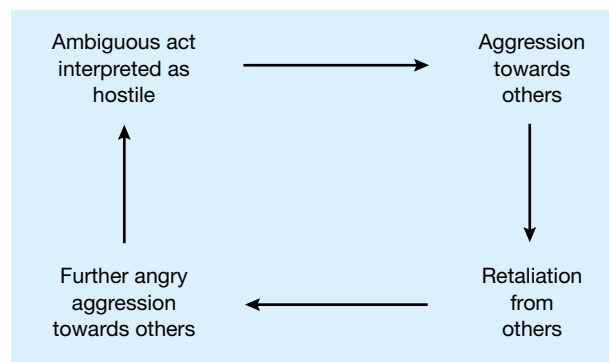
A social-cognitive perspective on aggressive behaviour (and, by extension, conduct disorder) comes from the work of Kenneth Dodge and associates. In one of his early studies (Dodge & Frame, 1982), Dodge found that the social information processing of aggressive children had a particular bias; these children interpreted ambiguous acts, such as being bumped in line, as evidence of hostile intent. Such perceptions may lead these children to retaliate aggressively for actions that may not have been intended as provocative. This can create a vicious cycle: their peers, remembering these aggressive behaviours, may tend to be aggressive more often against them, further angering the already aggressive children (see figure 10.3). More recently, Dodge and colleagues have linked deficits in social information processing to heart rate among adolescents who exhibit antisocial behaviour. Specifically, low heart rate predicted antisocial behaviour for male adolescents independent from social information processing deficits, a finding consistent with studies reviewed earlier on low arousal and conduct problems. However, the link between low heart rate and antisocial behaviour was accounted for by social information processing deficits for both male and female adolescents (Crozier, Dodge, Griffith et al., 2008).

### Peer influences

Investigations of how peers influence aggressive and antisocial behaviour in children have focused on two broad areas: (1) acceptance or rejection by peers and (2) affiliation with deviant peers. Studies have shown that being rejected by peers is causally related to aggressive behaviour, particularly in combination with ADHD (Hinshaw & Melnick, 1995). Other studies have shown that being rejected by peers can predict later aggressive behaviour, even after controlling for prior levels of aggressive behaviour (Coie & Dodge, 1998). Associating with other deviant peers also increases the likelihood of delinquent behaviour (Capaldi & Patterson, 1994).

**FIGURE 10.3**

Dodge's cognitive theory of aggression. The interpretation of ambiguous acts as hostile is part of a vicious cycle that includes aggression towards and from others.



Do children with conduct disorder choose to associate with like-minded peers, thus continuing on their path of antisocial behaviour (i.e., a social selection view), or does simply being around deviant peers help initiate antisocial behaviour (i.e., a social influence view)? Studies examining gene–environment interactions have shed light on this question, and the answer appears to be that both views are correct. That is, as we reviewed earlier, we know that genetic factors are at play in conduct disorder, and these factors in turn play a role in encouraging children with conduct disorder to select more deviant peers to associate with. However, environmental influence, particularly neighbourhood (e.g., poverty in the neighbourhood) and family (e.g., parental monitoring) factors play a role in whether children associate with deviant peers, and this in turn influences and exacerbates conduct disorder (Kendler, Jacobson, Myers, & Eaves, 2008).

## Treatment of conduct disorder

The treatment of conduct disorder appears to be most effective when it addresses the multiple systems involved in the life of a child (family, peers, school, neighbourhood).

### Family interventions

Some of the most promising approaches to treating conduct disorder involve intervening with the parents and families of the child. In addition, evidence suggests that intervening early, if even just briefly, can make an impact. In a randomised controlled trial (Shaw, Dishion, Supplee, Gardner, & Arnds, 2006), researchers compared what is called the family checkup (FCU) treatment to no treatment. FCU involves three meetings to get to know, assess, and provide feedback to parents regarding their children and parenting practices. In this study, FCU was offered to families with toddlers who were at high risk of developing conduct problems (based on the presence of conduct or substance abuse problems in parents or early signs of conduct behaviour in the children). This brief, three-session intervention was associated with less disruptive behaviour compared to no treatment, even two years after the intervention.

Gerald Patterson and colleagues have worked for over four decades developing and testing a behavioural program called **parent management training (PMT)**, in which parents are taught to modify their responses to their children so that prosocial rather than antisocial behaviour is consistently rewarded. Parents are taught to use techniques such as positive reinforcement when the child exhibits positive behaviours and time-out and loss of privileges for aggressive or antisocial behaviours.

This treatment has been modified by others, but, in general, it is the most efficacious intervention for children with conduct disorder and oppositional defiant disorder. Both parents' and teachers' reports of children's behaviour and direct observation of behaviour at home and at school support the program's effectiveness (Kazdin, 2005; Patterson, 1982). PMT has been shown to alter parent-child interactions, which in turn is associated with a decrease in antisocial and aggressive behaviours (Dishion & Andrews, 1995; Dishion, Patterson, & Kavanagh, 1992).

Parent and teacher training approaches have been incorporated into larger community-based programs and have been shown to reduce childhood conduct problems and increase positive parenting behaviours (Webster-Stratton, 1998; Webster-Stratton, Reid, & Hammond, 2001).

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Parent management training can be effective in treating conduct disorder.



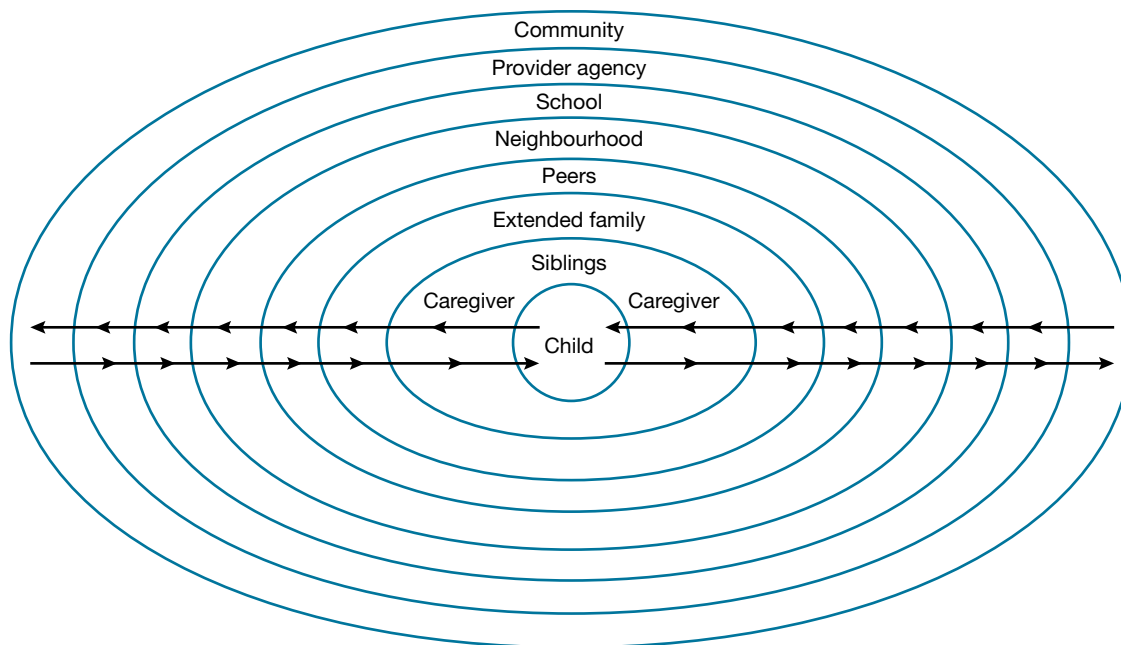
### Multisystemic treatment

Another promising treatment for serious juvenile offenders is **multisystemic treatment (MST)** (Borduin et al., 1995). MST involves delivering intensive and comprehensive therapy services in the community, targeting the adolescent, the family, the school and, in some cases, the peer group (figure 10.4). The treatment is based on the view that conduct problems are influenced by multiple factors within the family as well as between the family and other social systems.

The strategies used by MST therapists are varied, incorporating behavioural, cognitive, family-systems and case-management techniques. The therapy's uniqueness lies in emphasising individual and family strengths, identifying the social context for the conduct problems, using present-focused and action-oriented interventions, and using interventions that require daily or weekly efforts by family members. Treatment is provided in 'ecologically valid' settings, such as the home, school or local recreational centre, to maximise the chances that improvement will carry through into the regular daily lives of children and their families. MST has been shown to be effective in a number of studies (Henggeler, Schoenwald, Borduin, Rowland, & Cunningham, 1998; Ogden & Halliday-Boykins, 2004).

**FIGURE 10.4**

Multisystemic treatment (MST) includes consideration of many different factors when developing a child's treatment, including family, school, community and peers.



### Prevention programs

It would be ideal if we could prevent conduct disorder from ever developing. Can this be done? One such prevention program that has been studied for 20 years is called Fast Track. Findings from the Conduct Problems Prevention Research Group (CPRG), the group of researchers who developed, implemented and evaluated Fast Track, point to just how difficult it is to prevent something as complex as conduct disorder.

In this study, nearly 10 000 kindergarten children from four poor, high-crime regions in the United States were evaluated. From this large group, nearly 900 children who were exhibiting conduct problems were randomly assigned to either the Fast Track intervention or a control intervention. The Fast Track intervention was designed to help children academically, socially and behaviourally, focusing on areas that are problematic in conduct disorder, including peer relationships, aggressive and disruptive

behaviour, social information processing, and parent–child relationships. The intervention was delivered over the course of 10 years in groups and at individual families’ homes, with a more intensive treatment in years 1 to 5 and less intensive in years 6 to 10.

Results from the study showed that children who received the Fast Track intervention benefited. However, the benefits appeared to dwindle as the children got older. For example, children who received Fast Track displayed fewer behaviour problems and better social information processing skills than children in the control condition at the end of first grade, but by third grade some of the gains in social information processing were no longer evident, and even fewer gains were apparent by fourth and fifth grade (Conduct Problems Prevention Research Group, 2010a).

However, the news is not all bad. The intervention was still making a difference in some domains. By ninth grade, the children who had the most severe problem behaviours at the baseline assessment in kindergarten and who received Fast Track exhibited fewer delinquent behaviours and were less likely to have a diagnosis of conduct disorder or any externalising disorder compared to children in the control group (Conduct Problems Prevention Research Group, 2010a, 2011). Results from an additional analysis of outcomes in ninth grade revealed that the impact of Fast Track on reducing behaviour problems was due, in part, to a decrease in the hostile attribution bias discussed earlier (Dodge & Godwin, 2013). By the end of high school, a full two years after the intervention ended, children who received Fast Track were less likely to have been arrested (Conduct Problems Prevention Research Group, 2010b) and receive a diagnosis of any externalising disorder (Conduct Problems Prevention Research Group, 2011).

## 10.3 Depression and anxiety in children and adolescents

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**LEARNING OUTCOME 10.3** Discuss the description, aetiology and treatments for internalising disorders, including depression and anxiety disorders.

So far, we have discussed disorders that are specific to children. The internalising disorders, which include depression and anxiety disorders, first begin in childhood or adolescence but are quite common in adults as well. Much richer descriptions of these disorders are presented in the chapter on mood disorders and the chapter on anxiety disorders, obsessive-compulsive and trauma-related disorders. Here, we describe the ways in which the symptoms, aetiology and treatment of these disorders differ in children as compared to adults.

### Depression

#### Clinical descriptions, prevalence, and comorbidities of depression in childhood and adolescence

There are both similarities and differences in the symptomatology of children and adults with major depressive disorder (Garber & Flynn, 2001). Children and adolescents ages 7 to 17 and adults show the following symptoms: depressed mood, inability to experience pleasure, fatigue, concentration problems and suicidal ideation. Children and adolescents differ from adults in showing more guilt but lower rates of early-morning wakefulness, early-morning depression, loss of appetite and weight loss. As in adults, depression in children is recurrent. Longitudinal studies have demonstrated that both children and adolescents with major depression are likely to continue to exhibit significant depressive symptoms when assessed even four to eight years later (Garber, Kelly, & Martin, 2002; Lewinsohn, Rohde, Seeley, Klein, & Gotlib, 2000).

Depression occurs in 2 to 3 percent of school-age children under age 13 (Costello, Erkanli, & Angold, 2006). By adolescence, rates of depression range from 6 to 16 percent for girls and 4 to 7 percent for boys (Costello et al., 2006; Merikangas et al., 2010).

The prevalence among adolescent girls is almost twice that among adolescent boys, just as we have seen with adult depression (see focus on discovery 3.1). Although adolescent girls experience depression more often than adolescent boys, there are few differences in the types of symptoms they experience (Lewinsohn, Petit, Joiner, & Seeley, 2003). Interestingly, the gender difference does not occur before age 12; the full gender difference does not emerge until adolescence (Hankin et al., 1998).

As with adults, depression is comorbid with anxiety, particularly in adolescents (Cummings, Caporino, & Kendall, 2014). There is recent evidence that comorbidity between depression and anxiety in adolescence may be caused in part by shared genetic vulnerabilities (Waszczuk, Zavos, Gregory, & Eley, 2014).

### **Aetiology of depression in childhood and adolescence**

What causes a young person to become depressed? As with adults, evidence suggests that genetic factors play a role (Klein, Lewinsohn, Seeley, & Rohde, 2001). Indeed, the results of genetic studies with adults (see the chapter on mood disorders) apply to children and adolescents also, since genetic influences are present from birth, though they may not be expressed right away. A child with a depressed parent has four times the risk of developing depression as a child without a depressed parent (Hammen & Brennan, 2001). Of course, having a depressed parent likely confers risk via both genes and environment.

As has been found with older adults (see the chapter on mood disorders), gene–environment interactions predict the onset of depression in late adolescence and early adulthood. Results from the Northwestern-UCLA Youth Emotion Project, a large prospective study beginning with adolescents in their junior year of high school, are informative with respect to such interactions. In this study, those individuals who had a short allele of the serotonin transporter gene (short-short or short-long; see chapter 1) *and* had experienced significant interpersonal stressful life events were more likely to have a major depressive disorder episode than either those people who had a short allele but no such stress or those people who experienced interpersonal stressful life events but had the long-long allele combination of the gene (Vrshek-Schallhorn, Mineka, Zinbarg et al., 2013). Other research shows that interpersonal factors seem to be particularly important in predicting the development of depression among adolescent girls (Hammen, 2009).

As with adults, other types of early adversity and negative life events also play a role (Garber, 2006). For example, one study found that early adversity (e.g., financial hardship, maternal depression, chronic illness as a child) predicted depression between ages 15 and 20, particularly among adolescents who had experienced a number of negative life events by age 15 (Hazel, Hamman, Brennan, Najman, 2008). Rejection by parents is modestly associated with depression in childhood, as confirmed by a meta-analysis of 45 studies (McLeod, Wood, & Weisz, 2007). The effect size for parental rejection was considered small across the studies, suggesting that factors other than parental rejection play a larger role in causing depression in childhood.

As we learned in chapter 1, our bodies respond to stress via the HPA axis and the release of cortisol. Additional results from the Youth Emotion Project study indicate that cortisol taken first thing in the morning prospectively predicted the onset of a major depressive episode up to two and a half years later (Vrshek-Schallhorn, Doane, Mineka et al., 2013). These results are consistent with findings from adults (reviewed in the chapter on mood disorders).

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Many of the symptoms of childhood depression are the same as adult depression, including sad mood.



Recall from discussions in the chapter on mood disorders that cortisol in people with depression is associated with smaller volumes (size) of the hippocampus. This may also be true for adolescents. A longitudinal study of adolescents at risk for developing depression found that the volume of the hippocampus grew more slowly between early and middle adolescence for those who developed an episode of depression compared to those who did not develop an episode (Whittle et al., 2014). Although these researchers did not measure stressful life events or cortisol, the evidence from the different studies reviewed here suggests that genes, stressful life events, cortisol, and the brain are all important when considering depression in adolescence.

Consistent with both Beck's theory and the hopelessness theory of depression (see the chapter on mood disorders), cognitive distortions and negative attributional styles are associated with depression in children and adolescents in ways similar to what has been found with adults (Garber et al., 2002; Lewinsohn et al., 2000). For example, research with children with depression indicates that their outlooks are more negative than are those of children without depression and resemble those of adults with depression (Prieto, Cole, & Tageson, 1992). Negative thoughts and hopelessness also predict a slower time to recovery from depression among adolescents (Rhode, Seeley, Kaufman, Clarke, & Stice, 2006). Of course, depression can make children think more negatively (Cole, Martin, Peeke, Seroczynski, & Hoffman, 1998). Hence, it is important to consider longitudinal research.

A key question in the study of children with depression is: when do children actually develop stable attributional styles? That is, can young children have a stable way of thinking about themselves in the midst of such profound cognitive development? A longitudinal study examined the development of attributional style in children (Cole, Ciesla, Dallaire et al., 2008). Specifically, the researchers prospectively studied three groups of children for four years each. At year 1 of the study, the three groups were (1) children in second grade, (2) children in fourth grade, and (3) children in sixth grade. These three groups were followed yearly until the children were in grades 5, 7 and 9, respectively. The researchers found that attributional style didn't appear to be a stable style until children were early adolescents. In addition, attributional style did not interact with negative life events to predict depression (i.e., it was not a cognitive diathesis) for young children. It wasn't until the children were in eighth or ninth grade that support for attributional style as a cognitive diathesis emerged. Thus, results of this study suggest that attributional style becomes style-like by early adolescence and serves as a cognitive diathesis for depression by the middle school years.

#### RESEARCH EXAMPLE

##### Depression in Indigenous children in Victoria

Depression is a pervasive mental health problem across Australia. The prevalence of depression in children and adolescents in Australia is relatively high and typically three times higher among Aboriginal youth (Adams, Halacas, Cincotta, & Pesich, 2014). In addition, rates of depression affect more young females than males and this typically continues from childhood through to adolescence and into adulthood (Naylor, 2009).

Currently, there is an over-representation of calls from Indigenous children in Victoria to Kids Helpline for suicide and self-harm reasons (Adams et al., 2014). As research suggests, depression is known to be associated with significant impairment in various social domains (ABS, 2015), which makes daily activities such as development of friendships, seeking and obtaining employment and participating in sporting and/or extracurricular curricular activities difficult.

On a positive note, Aboriginal and Torres Strait Islander people with strong supportive networks and links to community regularly report lower levels of psychological distress (Commonwealth of Australia, 2013), which could ultimately decrease the prevalence of depression within the community. As there is evidence that early depressive episodes recur and persist into adult life along with ongoing psychosocial difficulties (Naylor, 2009), it is recommended that mental health social and psychological support

networks align practice to be culturally sensitive and age appropriate. *The Victorian Public Health and Wellbeing Plan 2015–2019* is an example of additional preventative training and professional development for people who work in the field of mental health; it incorporates supportive networks and links to the community as a strategic and manageable plan (Victorian State Government, 2015).

#### QUESTION

Explain the additional considerations you might need to make when diagnosing and treating an Indigenous child or adolescent with depression.

### Treatment of childhood and adolescent depression

Results from a large randomised controlled trial called the Treatment for Adolescents with Depression Study (TADS) provide some support for the efficacy of antidepressants. In the TADS study, adolescents were randomised to receive either Prozac, cognitive–behavioural therapy (CBT), or both combined. Results indicated that the combined treatment was the most effective through 12 weeks and that Prozac had modest advantages compared to CBT (March, Silva, Petrycki et al., 2004), a pattern that remained true after 36 weeks (TADS team, 2007). A meta-analysis of 27 randomised controlled trials of antidepressant medication treatment for depression and anxiety disorders in children found that the medications were most effective for anxiety disorders other than obsessive compulsive disorder (OCD) and less effective for OCD and depression (Bridge, Iyengar, Salary et al., 2007).

However, several concerns have been raised about antidepressants (see focus on discovery 10.2). The side effects experienced by some children taking antidepressants include diarrhoea, nausea, sleep problems and agitation (Barber, 2008). More importantly, concerns with respect to suicidality prompted a series of hearings in the United States and the United Kingdom about the safety of antidepressants for children. In the study cited above (March et al., 2004), 7 out of 439 adolescents attempted suicide, of whom 6 were in the Prozac group and 1 was in the CBT group. (See focus on discovery 10.2 for more discussion of this complex issue.) In the meta-analysis by Bridge and colleagues (2007), the researchers looked at suicidality rates in the studies of depression. The risk of suicidal ideation was 3 percent for those children taking antidepressants and 2 percent for those taking placebo. It is important to note that this analysis shows that children taking medication were at risk for suicidal ideation, not that medication caused the suicide thoughts or attempts. There were no completed suicides in any of the 27 studies reviewed.

Another issue has to do with how long the treatment effects last. A naturalistic follow-up of just under half the adolescents in the TADS study found that although most (96 percent) had recovered two years after the study, close to half of those who recovered by the end of treatment had a recurrent episode of depression five years later (Curry et al., 2011). Girls were more likely to have a recurrence than boys, as were adolescents who had a comorbid anxiety disorder. However, the rate of recurrence did not differ depending on the kind of treatment the adolescents received during the TADS study. In other words, the modest benefits of Prozac over cognitive–behavioural therapy reported in the TADS study did not seem to protect this group of adolescents from having a future episode of depression five years later.

Cognitive–behavioural therapy (CBT) in school settings appears to be effective and is associated with more rapid reduction of symptoms than family or supportive therapy (Curry, 2001). About 63 percent of adolescents with depression treated with CBT show significant improvement at the end of treatment (Lewinsohn & Clarke, 1999). However, other evidence suggests that the benefits of CBT may not last long for young people (Weisz, McCarty, & Valeri, 2006). Some evidence shows that CBT is most beneficial for Caucasian adolescents, those adolescents with good coping skills at pretreatment, and adolescents with recurrent depression (Rhode et al., 2006). The clinical case of Sharon illustrates CBT techniques with an adolescent.

## CLINICAL CASE

### Sharon

When initially seen, Sharon was extremely dysphoric, experienced recurrent suicidal ideation, and displayed a number of vegetative signs of depression ... [After being] placed on antidepressant medication ... she was introduced to a cognitive-behavioural approach to depression ... She was able to understand how her mood was affected by her thoughts and behaviour and was able to engage in behavioural planning to increase the occurrence of pleasurable and mastery-oriented events. Sharon manifested extremely high standards for evaluating her performance in a number of areas, and it became clear that her parents also subscribed to these standards, so that family therapy sessions were held to encourage Sharon and her parents to re-evaluate their standards.

Sharon had difficulty with the notion of changing her standards and noted that when she was not depressed she actually valued her perfectionism. At that point she resisted the therapy because she perceived it as trying to change something she valued in herself. With this in mind, we began to explore and identify those situations or domains in which her perfectionism worked for her and when and how it might work against her. She became increasingly comfortable with this perspective and decided she wanted to continue to set high standards regarding her performance in mathematical course work (which was a clear area of strength), but she did not need to be so demanding of herself regarding art or physical education. (Adapted from Braswell & Kendall, 1988, p. 194)

### QUESTIONS

1. How has CBT affected Sharon's life?
2. Would you have considered medication for Sharon? Why or why not?

A good deal of work has focused on how to prevent the onset of depression in adolescents and children. A meta-analysis examined two types of preventive interventions for depression: selective and universal (Horowitz & Garber, 2006). Selective prevention programs target particular youth based on family risk factors (e.g., parents with depression), environmental factors (e.g., poverty) or personal factors (e.g., hopelessness). Universal programs are targeted towards large groups, typically in schools, and seek to provide education and information about depression. Results of the meta-analysis indicated that selective prevention programs were more effective than universal programs in preventing depression symptoms among adolescents.

A large randomised control clinical trial of a selective prevention program for at-risk adolescents, defined as having at least one parent with depression, showed promising results (Garber et al., 2009). Adolescents were randomly assigned to a group CBT intervention that focused on problem-solving skills and changing negative thoughts or to a usual care group (i.e., any mental health care they sought out on their own). The incidence of depression episodes was lower for adolescents in the CBT group than for those in the usual care group; therefore, the treatment may have had an effective preventative effect.

## Anxiety

Just about every child experiences fears and worries as part of the normal course of development. Common fears, most of which are outgrown, include fear of the dark and of imaginary creatures and fear of being separated from parents. In general, as with adults, fears are reported more often for girls than for boys (Lichenstein & Annas, 2000), though this sex difference may be due at least in part to social pressures on boys that make them reluctant to admit that they are afraid of things.

The seriousness of some childhood anxiety problems should not be underestimated. Not only do children suffer, as do adults, from the aversiveness of being anxious — simply put, anxiety doesn't feel good — but their anxiety may also work against their acquisition of skills appropriate to various stages of their development. For example, a child who is painfully shy and finds interacting with peers virtually

intolerable is unlikely to learn important social skills. This deficit may persist as the child grows into adolescence and will form the foundation of still further social difficulties. Then, whether in the workplace or at college, the adolescent's worst fear — 'people will dislike and reject me' — is likely to be realised as his or her awkward, even off-putting, behaviour towards others may produce rejecting and avoiding responses.

### **Clinical descriptions and prevalence of anxiety in childhood and adolescence**

For fears and worries to be classified as disorders according to DSM criteria, children's functioning must be impaired; unlike adults, however, children need not regard their fear as excessive or unreasonable, because children sometimes are unable to make such judgements. Based on these criteria, about 3 to 5 percent of children and adolescents would be diagnosed as having an anxiety disorder (Rapee, Schniering, & Hudson, 2009). Using data from the National Comorbidity Replication study, researchers found that over 30 percent of the 10 000 adolescents ages 13 to 18 who were interviewed had a lifetime occurrence of an anxiety disorder. Specific phobias (19.3 percent) and social anxiety disorder (9.1 percent) were the most common.

**Separation anxiety disorder** in childhood is characterised by constant worry that some harm will befall their parents or themselves when they are away from their parents. When at home, such children shadow one or both of their parents. Since the beginning of school is often the first circumstance that requires lengthy and frequent separations of children from their parents, separation anxiety is often first observed when children begin school.

Anxiety disorders in childhood and adolescence can interfere with other aspects of development.



#### **FOCUS ON DISCOVERY 10.2**

### **Controversies in the diagnosis and treatment of children with psychopathology**

The number of children diagnosed with psychological disorders continues to rise, sometimes dramatically, as has the number of children taking psychoactive medications. These increases raise several questions.

- Has there truly been an increase in the number of children with psychological disorders?
- Have our diagnostic system and assessment measures improved enough to identify children once overlooked?
- Are children being misdiagnosed and then treated for problems they do not have?
- Are medications safe for children?
- Will medication use lead to later drug use or abuse among children?

Here we briefly discuss some of the controversies and current evidence accumulated to address these questions.

#### **Bipolar disorder in children**

For years, professionals thought bipolar disorders were very rare or even non-existent among children. Today, however, diagnoses of bipolar disorders in children have increased dramatically. Has bipolar disorder among children increased? Probably not. In fact, bipolar disorder in children is much higher in the United States than in other countries, suggesting that the increase is specific to the United States (James et al., 2014; Van Meter, Moreira, & Youngstrom, 2011).

One of the difficult diagnostic issues facing mental health professionals is distinguishing bipolar disorders from ADHD. Agitated behaviour can be a sign of both, and only through careful and thorough assessments can the distinction be made. An early controversy was whether the diagnostic criteria for bipolar disorders in children should be the same as the criteria for bipolar disorders in adults. Some argued that the criteria for children should include explosive but brief outbursts of emotion and behavioural dysregulation (e.g., Biederman et al., 2000), but these are fundamentally different from the current DSM criteria for bipolar I disorder (see the chapter on mood disorders), and emotion dysregulation is also present in ADHD (Carlson & Meyer, 2006; Dickstein & Leibenluft, 2006). Later research has confirmed that the adult criteria are applicable to children and adolescents (Youngstrom, Freeman, & Jenkins, 2009). The American Academy of Child and Adolescent Psychiatry recommends using the adult DSM-5 criteria for diagnosing bipolar disorder in children and adolescents (McClellan, Kowatch, & Findling, 2007). These guidelines also recommend that impairment be identified in two different settings (e.g., home, school), a requirement not found in the DSM.

The DSM-5 added a new category called *disruptive mood dysregulation disorder* (DMDD) in the hopes that this will help clinicians distinguish emotion dysregulation that is part of bipolar disorder from severe irritability, thus reducing the number of children receiving a diagnosis of bipolar disorder. The criteria for DMDD include severe temper outbursts that tend to happen three times a week and in two different settings (e.g., home, school). The diagnosis is only for children between the ages of 6 and 18, and it must be diagnosed prior to age 10. The symptoms need to have been present for at least a year.

Because this category was only introduced in 2013, not much research has been done yet. In fact, it was added to DSM-5 without much research support to warrant its inclusion, and this has rightly been controversial (Axelson et al., 2011). Research on severe mood dysregulation, a category not in DSM-IV but developed by researchers to capture children who exhibited irritability and not mania, showed that children with such extreme irritability were also highly likely to meet the diagnostic criteria for ADHD or ODD. However, over the course of more than 2 years, only 1 out of 84 children with severe mood dysregulation developed a manic episode (Leibenluft, 2011). These findings support the inclusion of DMDD in DSM-5, at least with respect to distinguishing it from bipolar disorder. Unfortunately, the new disorder may be problematic with respect to its reliability: the DSM-5 field trials (see the chapter on diagnosis and assessment) found that the reliability of the DMDD criteria was poor (Regier et al., 2013). This does not bode well for the new category.

One study sought to assess the DMDD criteria using data from three ongoing longitudinal studies of childhood psychopathology (Copeland, Angold, Costello, & Egger, 2013). Even though these studies began before the DMDD criteria were released in May 2013, the researchers were able to test the DMDD criteria in these samples because 'its criteria overlap entirely with those of other common disorders' (p. 174). Such overlap in criteria suggests that the new diagnosis will be comorbid with other conditions. Indeed, this study found significant comorbidity between DMDD and depression, anxiety, ODD and ADHD. Although many childhood disorders are comorbid with one another, the comorbidity between DMDD and ODD was very high, suggesting that these might not be distinct categories. One of the seven children who had a manic episode also met the criteria for DMDD. Additional research in the coming years will reveal whether or not DMDD is a category that should remain in the DSM.

### **Antidepressant medications**

Can antidepressant medications increase the likelihood of suicide in adolescents? This question was the focus of intense debate 10 years ago, and the issue is still not fully resolved.

Findings from the TADS (Treatment of Adolescent Depression Study) indicated that the most effective treatment was a combination of Prozac and cognitive-behavioural therapy (March et al., 2004). However, the authors also reported that six adolescents taking Prozac attempted suicide (1.5 percent of the sample), whereas only one receiving CBT attempted suicide. The participants in the study were randomly assigned to treatment conditions, so it is less likely that the adolescents taking Prozac were more seriously ill or suicidal than the ones receiving CBT.

Antidepressants can take as long as three to four weeks to start working (see the chapter on mood disorders), and one analysis of adolescent suicide attempts and antidepressant use found that the risk for suicide was highest in the first three to four weeks of treatment. Thus, it could be the case that the medications did not have sufficient time to begin working in the adolescents who attempted suicide. It may also be true that the combined treatment in the TADS was most effective because CBT began working earlier in the course of treatment.

These findings prompted the Food and Drug Administration (FDA) in the United States to hold a series of hearings on the safety of treating children and adolescents with antidepressant medications. The United Kingdom had already made a strong statement that the benefits of antidepressants for the treatment of adolescent depression were not greater than the risks. At the end of the FDA hearings, the panel mandated a 'black box' warning to accompany information sent to physicians on the use of antidepressants with adolescents. This is the strongest safety warning the FDA can issue with medications. In the United Kingdom, the equivalent regulatory agency, the Medicines and Healthcare Products Regulatory Agency (MHRA), also recommended warnings for antidepressant labels. Since then, the numbers of prescriptions for antidepressants have declined, in the United States and in the United Kingdom (Kurian et al., 2007). Currently, no antidepressant is approved in Australia for use in children and adolescents (Adverse Drug Reactions Advisory Committee, 2014). Although suicides among youths have decreased at the same time, too many other variables have changed during this time to know whether the reduction in antidepressant use explains the decline in suicide rates.

### **Stimulant medications**

As reviewed earlier, the number of children taking stimulant medications has risen dramatically. Does the use of stimulant medications lead to increases in illicit drug use among children? Three prospective, longitudinal studies suggest that the answer to this question is no. In one study, two groups of children with ADHD were studied for 13 years (Barkley, Fischer, Smallish, & Fletcher, 2003). One group of children had been treated with stimulant medication for three and a half years on average and the other group had never received stimulant medication. At follow-up in young adulthood, those who had taken stimulant medication were not more likely to have used illicit drugs than those who had not been treated, with one exception — those who had taken stimulant medication were at greater risk for having tried cocaine. However, after controlling for the severity of conduct disorder symptoms, the relationship between stimulant medication use and trying cocaine disappeared. This suggests that having severe conduct disorder symptoms accounts for the link between stimulant medication and trying cocaine, not the use of stimulant medication per se.

The second study followed into adulthood a group of children with reading disorders who had been treated with stimulant medications for 12 to 18 weeks and compared them to a group of children with reading disorders who had not received stimulant medication. Sixteen years after the medication treatment, the two groups did not differ in their use of illicit drugs (Mannuzza, Klein, & Moulton, 2003).

The third study followed up children eight years after they participated in the MTA treatment study (discussed earlier) when they were in middle to late adolescence (Molina et al., 2013). Children with ADHD, regardless of treatment type, were more likely to have used alcohol, tobacco, marijuana and other drugs in adolescence than the children without ADHD, a finding that is consistent with studies showing comorbidity between ADHD and substance use. However, there was no relationship between stimulant medication use and later drug use. Neither the total amount of medication taken, the dosage of medication, or initial MTA treatment group (medication alone, combined treatment), or continued use of medication after the MTA study was associated with later drug or alcohol use. Thus, children with ADHD are at a higher risk for substance use, but it is not because they have taken stimulant medication.

### **Autism spectrum disorder: diagnosis and causes**

The number of cases of autism spectrum disorder (ASD) has increased dramatically over the past 15 years. In New Zealand, there are approximately 77 500 people, or 2 percent of the population with ASD (Autism NZ, n.d.). As reported by the Australian Bureau of Statistics (ABS), the prevalence rate of ASD in Australia as of 2012 is an estimated 0.5 percent of the population. This has risen by 79 percent from 2009 (ABS, 2012). Why has there been such an increase? Are there that many more children with autism, or have mental health professionals gotten better at making a diagnosis?

Autism wasn't formally recognised in the DSM until 1980, and the diagnostic criteria broadened quite a bit between the publication of DSM-III in 1980 and the release of DSM-IV in 1994. (Asperger's disorder was first formally recognised in DSM-IV; this is now combined with autism in DSM-5.) More children met the criteria for a diagnosis of autism under the broader criteria of DSM-IV than they did under the narrower criteria of DSM-III (Gernsbacher, Dawson, & Goldsmith, 2005). Additionally, there is greater public awareness of ASD, and this may spur families to seek out mental health professionals for a formal psychological assessment. Indeed, the delay in or lack of language acquisition has become a widely

recognised warning sign among parents and mental health professionals that ASD may be a consideration. In addition, public schools are mandated by law to provide services for children with ASD, and this may have helped families to seek a formal diagnosis. Although the rise in autism diagnoses may be accounted for in part by better diagnosis, awareness and mandated services, most experts agree that there are actually more cases today than there were 30 years ago.

With the increase in ASD have come concerns over what might be causing this increase. Based largely on celebrity proclamations, parents became particularly worried that vaccines routinely given to toddlers may cause ASD. The MMR vaccine (for measles, mumps and rubella) is given to children right around the age when autism signs and symptoms begin to appear. A related concern is that the product used to preserve these vaccines — a substance called thimerosal, which contains mercury — may be responsible for autism. This absurd and incorrect link between vaccinations and ASD was brought into public vernacular when Dr AJ Wakefield's fraudulent paper was published in a 1998 edition of *The Lancet* (Wakefield et al., 1998). There is no evidence linking autism with either the MMR vaccine or thimerosal. Vaccines have not been stored in thimerosal for the last several years, and even those vaccines that were stored in thimerosal contained very small amounts of mercury. One study examined the number of ASD diagnoses reported to the California Department of Developmental Services between 1995 and 2007 (Schechter & Grether, 2008). By 2001, all but the smallest trace of thimerosal had been removed from childhood vaccines. If thimerosal was causing autism, the decline in its use in vaccines might correspond to a decrease in the number of new cases of autism. However, the study found no such association. In fact, the number of new cases of autism increased. In May 2004, the Institute of Medicine published the results of its comprehensive review of available evidence on the link between MMR and autism. This report concluded that the MMR vaccines are not responsible for autism (Institute of Medicine, 2004).

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### QUESTIONS

1. Despite being disproven time and time again, why do you think there are some people who still incorrectly believe there is a link between ASD and vaccines?
2. Why do you think the TGA does not support antidepressant use in adolescents or children in Australia?

Two changes happened with DSM-5. First, the category was moved into the anxiety disorders chapter, and second, the age of onset prior to age 18 requirement was removed. Thus, adults can now receive a diagnosis of separation anxiety disorder. For adults, the anxiety stems from separation from an attachment figure that is not necessarily a parent (e.g., a spouse).

Another anxiety disorder among children and adolescents is social anxiety disorder. Most classrooms include at least one or two children who are extremely quiet and shy. Often these children will play only with family members or familiar peers, avoiding strangers both young and old. Their social anxiety may prevent them from acquiring skills and participating in a variety of activities enjoyed by most of their peers, for they avoid playgrounds and stay out of games played by other children. Extremely shy children may refuse to speak at all in unfamiliar social circumstances, a condition called *selective mutism*.

Prevalence estimates for social anxiety disorder among children and adolescents range from 1 to 7 percent (Merikangas et al., 2010; Rapee et al., 2009). Higher prevalence rates are observed with adolescents, who have a more acute concern about the opinions of others than younger children do.

Children who are exposed to traumas such as chronic abuse, community violence and natural disasters may experience symptoms of post-traumatic stress disorder (PTSD) similar to those experienced by adults. As many as 5 percent of children meet criteria for PTSD (Merikangas et al., 2010). For children older than age six, these symptoms are organised into the same four broad categories discussed in the chapter on anxiety, obsessive-compulsive and trauma-related disorders: (1) intrusively reexperiencing the traumatic event, as in nightmares, flashbacks, or intrusive thoughts; (2) avoiding trauma-related situations or information and experiencing a general numbing of responses, as in feelings of detachment or anhedonia; (3) negative changes in cognitions or mood related to the traumatic event; and (4) increased arousal and reactivity, which can include irritability, sleep problems and hypervigilance.

### DSM-5 criteria for separation anxiety disorder

People with separation anxiety disorder experience excessive anxiety that is not developmentally appropriate about being away from people to whom one is attached, with at least three of the following symptoms that last for at least four weeks (for adults, symptoms must last for six months or more):

- repeated and excessive distress when separated
- excessive worry that something bad will happen to an attachment figure
- refusal or reluctance to go to school, work, or elsewhere
- refusal or reluctance to sleep away from home
- nightmares about separation from attachment figure
- repeated physical complaints (e.g., headache, stomach ache) when separated from attachment figure.

DSM-5 added separate criteria for PTSD in children age six and younger. The symptoms for these younger children fall into the same four broad categories just described, but they are presented in ways that are more developmentally appropriate for younger children. For example, extreme temper tantrums are an example of an increased reactivity symptom; intrusive thoughts about the trauma may be experienced as re-enactment play. In addition, some of the symptom descriptors that don't apply to young children are removed from these criteria. For example, holding negative beliefs about oneself is part of the negative changes in cognitions or mood symptom cluster that does not apply to very young children.

Obsessive compulsive disorder (OCD) is also found among children and adolescents, with prevalence estimates ranging from less than 1 to 4 percent (Rapee et al., 2009). The symptoms in childhood are similar to symptoms in adulthood: both obsessions and compulsions are involved. The most common obsessions in childhood involve dirt or contamination as well as aggression; recurrent thoughts about sex or religion become more common in adolescence (Turner, 2006). OCD in children is more common in boys than girls, but by adulthood, OCD is slightly more common in women than men.

### Aetiology of anxiety disorders in childhood and adolescence

As with adults, genetics plays a role in anxiety among children, with heritability estimates ranging from 29 to 50 percent in one study (Lau, Gregory, Goldwin, Pine, & Eley, 2007). However, genes do their work via the environment, with genetics playing a role in separation anxiety in the context of more negative life events experienced by a child (Lau et al., 2007).

Parenting practices play a small role in childhood anxiety. Specifically, parental control and over-protectiveness, more than parental rejection, is associated with childhood anxiety. However, parental control accounted for only 4 percent of the variance in childhood anxiety according to a meta-analysis of 47 studies (McLeod, Weisz, & Wood, 2007). Thus, 96 percent of the variance is accounted for by other factors. Other psychological factors that predict anxiety symptoms among children and adolescents include emotion-regulation problems and insecure attachment in infancy (Bosquet & Egeland, 2006).

Theories of the aetiology of social anxiety in children are generally similar to theories of social anxiety in adults. For example, research has shown that children with anxiety disorders overestimate the danger in many situations and underestimate their ability to cope with them (Boegels & Zigterman, 2000). The anxiety created by these cognitions then interferes with social interaction, causing the child to avoid social situations and thus not to get much practice at social skills. In adolescence, peer relationships are important. Specifically, a longitudinal study found that adolescents who perceived that their peers did not accept them were more likely to be socially anxious (Teachman & Allen, 2007). Other research points to behavioural inhibition as an important risk factor for developing social anxiety (also discussed in the chapter on anxiety). Children who had high levels of behavioural inhibition at age 4 were 10 times as likely as children with lower levels to have social anxiety disorder by age 9 (Essex, Klein, Slattery, Goldsmith, & Kalin, 2010).

Theories about the causes of PTSD are similar for children and adults. For both, there must be exposure to a trauma, either experienced or witnessed. Like adults, children who have a propensity to experience anxiety may be at more risk for developing PTSD after exposure to trauma. Specific risk factors for children may include level of family stress, coping styles of the family, and past experiences with trauma (Martini, Ryan, Nakayama, Ramenofsky, 1990). Some theorists suggest that parental reactions to trauma can help to lessen children's distress; specifically, if parents appear to be in control and are calm in the face of stress, a child's reaction may be less severe (Davis & Siegal, 2000).

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Separation anxiety disorder involves an intense fear of being away from parents or other attachment figures.



### **Treatment of anxiety in childhood and adolescence**

For the most part, treatment of anxiety is similar to that employed with adults, with suitable modifications to accommodate the different abilities and circumstances of childhood. The major focus of

these treatments is on exposure. Compared to exposure treatments for adults, treatments may be modified for children by including more modelling (seeing an adult approach the feared object) and more reinforcement.

Evidence from a meta-analysis of 48 randomised controlled trials indicates that cognitive-behavioural therapy can be helpful to many children with anxiety disorders (Reynolds, Wilson, Austin, & Hooper, 2012). One of the more widely used treatments is called the Coping Cat (Kendall, Aschenbrand, & Hudson, 2003). This treatment focuses on confrontation of fears, development of new ways to think about fears, exposure to feared situations and relapse prevention. Parents are also included in a couple of sessions. Data from randomised controlled clinical trials have shown this treatment to be effective in the short term, seven years later (Kendall et al., 1997; Kendall, Safford, Flannery-Schroeder, & Webb, 2004), and 19 years later (Benjamin, Harrison, Settipani, Brodman, & Kendall, 2013).

Another randomised controlled trial compared individual CBT, family CBT and family psychoeducation for the treatment of childhood anxiety. Both individual and family CBT included the Coping Cat workbook, both were more effective than family psychoeducation at reducing anxiety (Kendall, Hudson, Gosch, Flannery-Schroeder, & Suveg, 2008) and the effects persisted at one-year and seven-year follow-up (Benjamin et al., 2013). The family CBT was more effective than individual CBT when both parents had an anxiety disorder. This study points to the importance of considering not only the child's anxiety but also levels of parental anxiety when deciding on a treatment for childhood anxiety.

Another study examined the Coping Cat treatment alone and in combination with sertraline (Zoloft) for children with separation anxiety, general anxiety and social anxiety, and found that the combination treatment was more effective than either the Coping Cat or medication alone (Walkup et al., 2008). Two- and three-year follow-up studies of these children found that children assigned to the Coping Cat alone or medication alone conditions continued to get better, such that these two conditions were as effective in reducing anxiety as the combination treatment (Piacentini et al., 2014). Thus, the combination treatment appears to provide the most immediate improvement, but over time the Coping Cat CBT treatment (and medication alone) yielded the same benefits.

Behaviour therapy and group cognitive-behavioural therapy have been found to be effective for social anxiety disorder in children (Davis, May, & Whiting, 2011). Only a few studies have examined the efficacy of cognitive-behavioural therapy for OCD in children and adolescents. In one study, CBT was equally as effective as medication, but CBT plus medication was more effective than medication alone but not more than CBT alone (O'Kearney, Anstey, & von Sanden, 2006). However, for children and adolescents with severe OCD, a combination of CBT and sertraline (Zoloft) was more effective than CBT alone (Pediatric OCD Treatment Study [POTS] team, 2004). In another study, CBT plus medication was more effective than medication alone or medication plus generic instructions about what CBT involves (Franklin et al., 2011). Results from a recent randomised controlled trial suggest that CBT is also effective for young children (i.e., ages five to eight). In this study, participants were randomly assigned to receive either family-based CBT, which included exposure plus response prevention (see the chapter on anxiety, obsessive-compulsive and trauma-related disorders), or family-based relaxation therapy. At the end of the 14-week treatment, children who received CBT had fewer symptoms and better functioning compared to the children who received the relaxation training (Freeman, Sapyta, Garcia et al., 2014).

Other methods of providing treatment, including 'bibliotherapy' and computer-assisted therapy, have shown promise as well. In bibliotherapy, parents are given written materials and are the 'therapist' with their children. Although this approach is effective in reducing childhood anxiety, it does not appear to be as effective as CBT group treatments (Rapee, Abbott, & Lyneham, 2006). Nevertheless, it will be important to develop these types of mobile treatments for people who live in areas where CBT therapists are not available or are too expensive.

Only a few studies have evaluated the efficacy of treatment of PTSD among children and adolescents, but the available research suggests that cognitive-behavioural treatments, whether individual or group, are effective for children and adolescents with PTSD (Davis, May, & Whiting, 2011).

## 10.4 Specific learning disorder

**LEARNING OUTCOME 10.4** Understand the learning disorders dyslexia and dyscalculia, and learn the causes and treatments for dyslexia.

A **specific learning disorder** is a condition in which a person shows a problem in a specific area of academic, language, speech or motor skills that is not due to intellectual disability or deficient educational opportunities. Children with a specific learning disorder are usually of average or above-average intelligence but have difficulty learning some specific skill in the affected area (e.g., arithmetic or reading), and thus their progress in school is impeded.

### CLINICAL CASE

#### Marcus

Marcus was excited about his psychology major and dreamed of becoming a clinical psychologist. He eagerly signed up for as many courses as would fit into his schedule and had open seats. He sat in the front of the large lecture courses, raising his hand and contributing to the discussion whenever possible. When it came time to take the first exam, he carefully reviewed his notes and textbook. He also experienced that creeping anxiety that came whenever he took written exams. Even though he thought he studied as much if not more than the other students, he just did not do well on the exams. 'He didn't test well' was what he told himself. But when he thought about it, he realised that when he was reading the textbook, the words and letters sometimes got scrambled in a way that made it difficult to remember the material.

When he received his grade on the first midterm, he was worried. It contained all sorts of marks from the grader indicating that his writing was indecipherable and that he had not covered all the concepts required for the correct answers. His graduate student instructor suggested that he go for testing at the campus clinic in charge of assessments for learning disorders.

After a comprehensive assessment from the campus clinic, Marcus received a diagnosis of specific learning disorder with the dyslexia specifier. Marcus was now going to get extra time for his exams and papers, and not just in his psychology classes but in all of his classes. The following semester, Tim's grade point average went up to a 3.8, and he felt confident that he was closer to achieving his goal of attending graduate school in clinical psychology.

#### QUESTIONS

1. What do you think the outcome would have been if Marcus had not gone for a comprehensive assessment? Give your reasoning.
2. Simply reviewing class notes did not seem to work for Marcus. What sorts of study activities might work better for him? Do some research online to support your answer.

### DSM-5

#### DSM-5 criteria for specific learning disorder

- People with specific learning disorder experience difficulties in learning basic academic skills (reading, mathematics, or writing) inconsistent with their age, schooling, and intelligence persisting for at least six months.
- People with specific learning disorder experience significant interference with academic achievement or activities of daily living.

## Clinical descriptions

The term *learning disabilities* is not used in DSM-5 but is used by mental health professionals to group together three categories that do appear in the DSM: specific learning disorder, communication

disorders, and motor disorders. These disorders are described briefly in table 10.3. Any of these disorders may apply to a child who fails to develop to the degree appropriate to his or her developmental level in a specific academic, language or motor skill area. These disorders are often identified and treated within the school system rather than through mental health clinics. An analysis of four large epidemiological samples indicates that a specific learning disorder involving reading is far more common in boys than in girls (Rutter, Caspi, Fergusson et al., 2004). The prevalence rates for specific learning disorders involving reading or maths (i.e., dyslexia and dyscalculia) are about the same, ranging from 4 to 7 percent of children (Landerl, Fussenegger, Moll, & Willburger, 2009).

**TABLE 10.3** Specific learning, communication and motor disorders in DSM-5

**Specific learning disorder** includes specifiers:

- **Dyslexia** (formerly called reading disorder) involves significant difficulty with word recognition, reading comprehension and typically written spelling as well.
- **Dyscalculia** (formerly called mathematics disorder) involves difficulty in producing or understanding numbers, quantities or basic arithmetic operations.

**Communication disorders** include the following:

- **Speech sound disorder** (formerly called phonological disorder) involves correct comprehension and sufficient vocabulary use, but unclear speech and improper articulation. For example, *blue* comes out *bu*, and *rabbit* sounds like *wabbit*. With speech therapy, complete recovery occurs in almost all cases, and milder cases may recover spontaneously by age eight.
- **Childhood onset fluency disorder** (stuttering) is a disturbance in verbal fluency that is characterised by one or more of the following speech patterns: frequent repetitions or prolongations of sounds, long pauses between words, substituting easy words for those that are difficult to articulate (e.g., words beginning with certain consonants) and repeating whole words (e.g., saying 'go-go-go-go' instead of just a single 'go'). DSM-IV-TR estimates that up to 80 percent of people with stuttering recover, most of them without professional intervention, before the age of 16.
- Language disorder (combines expressive and mixed receptive expressive language disorders from DSM-IV).
- Social (pragmatic) communication disorder (new in DSM-5).

**Motor disorders** include the following:

- Tourette's disorder involves one or more vocal and multiple motor tics (sudden, rapid movement or vocalisation) that start before the age of 18.
- Developmental coordination disorder (formerly called motor skills disorder) involves marked impairment in the development of motor coordination that is not explainable by intellectual disability or a disorder such as cerebral palsy.
- Stereotypic movement disorder involves seemingly purposeless movements repeated over and over that interfere with functioning and could even cause self-injury.

## Aetiology of specific learning disorder

Most research on specific learning disorder concerns dyslexia, perhaps because it is the most prevalent of this group of disorders: it affects 5 to 15 percent of school-age children. In DSM-5, neither dyslexia nor dyscalculia is named as a distinct disorder. Instead, dyslexia and dyscalculia are specifiers for the DSM-5 category specific learning disorder. Because more research has been done on dyslexia than on dyscalculia, we consider dyslexia here in some detail.

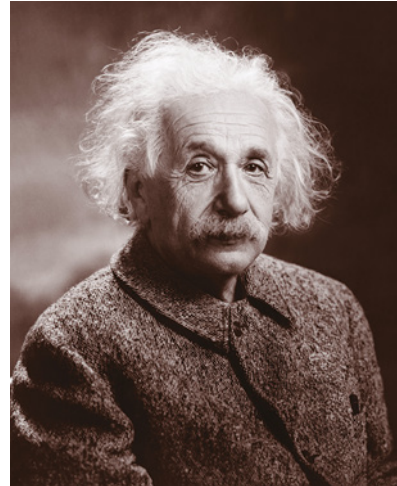
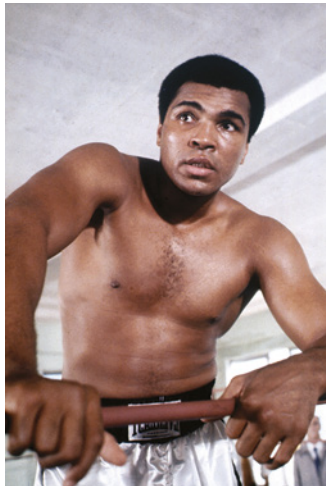
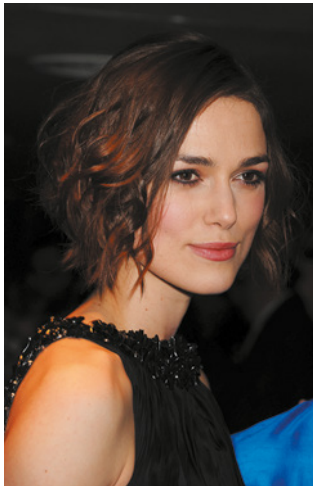
### Aetiology of dyslexia

Family and twin studies confirm that there is a heritable component to dyslexia (Pennington, 1995; Raskind, 2001). Furthermore, the genes that are associated with dyslexia are the same genes associated with typical reading abilities (Plomin & Kovas, 2005). These so-called generalist genes are thus important for understanding normal as well as abnormal reading abilities. Research has also examined

gene–environment interactions in dyslexia, and here the evidence so far suggests that the heritability of reading problems varies depending on parental education. Genes play a bigger role in dyslexia among children whose parents have more education compared to children whose parents have less education (Friend, DeFries, Olson, Pennington, 2009; Kremen et al., 2005). Homes with high parental education likely emphasise reading and provide a lot of opportunity for children to read. In this type of environment, then, a child’s risk for developing dyslexia is more driven by heritable combinations of genes than by environment.

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Many people have reached the top of their fields while living with a learning disability. Actor Keira Knightley and boxer Muhammad Ali were both diagnosed with dyslexia, while some believe Albert Einstein also had a learning disability.



Evidence from psychological, neuropsychological, and neuroimaging studies suggests that dyslexia involves problems in language processing. These problems include perception of speech and analysis of the sounds of spoken language and their relation to printed words (Mann & Brady, 1988), difficulty recognising rhyme and alliteration (Mann & Brady, 1988), problems with rapidly naming familiar objects (Scarborough, 1990; Wolf, Bally, & Morris, 1986), and delays in learning syntactic rules (Scarborough, 1990). Many of these processes fall under what is called *phonological awareness*, which is believed to be critical to the development of reading skills (Anthony & Lonigan, 2004).

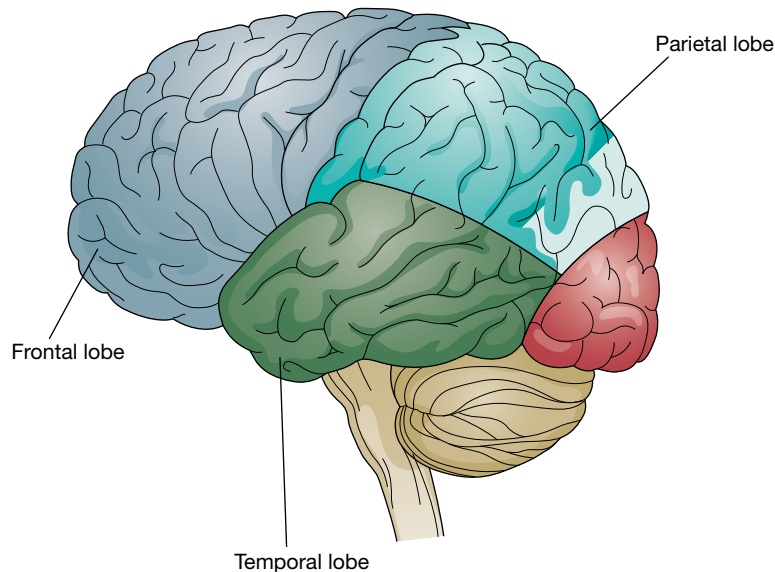
Early fMRI studies supported the idea that children with dyslexia have a problem in phonological awareness. These studies show that areas in the left temporal, parietal and occipital regions of the brain are important for phonological awareness, and these same regions are centrally involved in dyslexia (see figure 10.5).

For example, a study using fMRI found that, compared to children without dyslexia, children with dyslexia showed less activation in the left temporoparietal and occipitotemporal areas while doing a number of reading-relevant tasks, such as identifying letters and sounding out words (Shaywitz, Shaywitz, Pugh et al., 2002). A treatment study showed that after a year of intensive treatment for reading problems, children with dyslexia were better readers and also showed greater activation in the left temporoparietal and occipitotemporal areas while completing a reading task, compared to a group of children who received a less intensive treatment (Shaywitz, Shaywitz, Blachman et al., 2004).

More recent fMRI studies suggest that the problem might not be in areas of the brain that support phonological awareness per se, but in their connections to other areas of the brain that support the ability to produce speech, including Broca’s area (Boets et al., 2013; Vandermosten et al., 2012).

These findings suggest the interesting possibility that children with dyslexia may not have problems with phonological awareness but with integrating this awareness with generating the ability to read (Ramus, 2014). Future studies will need to be conducted to connect these recent fMRI studies with well-replicated neuropsychological studies revealing deficits in phonological awareness.

**FIGURE 10.5** Areas of the brain implicated in dyslexia include parts of the frontal, parietal and temporal lobes, at least for Western languages.



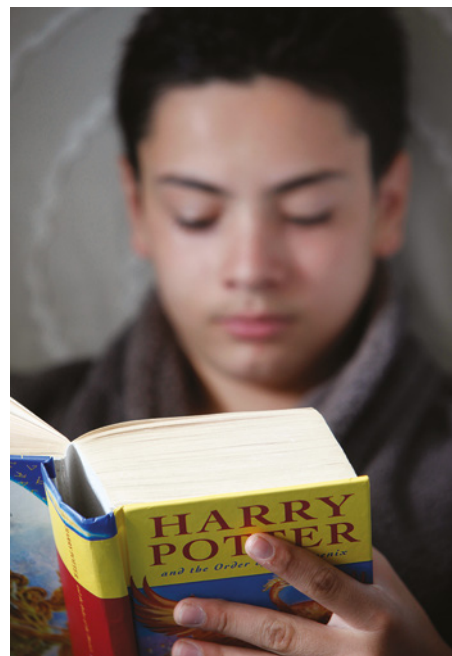
The fMRI studies discussed above involved participants in the United States who spoke English. A study examining Chinese children with dyslexia failed to find a problem with the temporoparietal area of the brain during reading tasks; instead, the left middle frontal gyrus showed less activation (Siok, Perfetti, Lin, & Tan, 2004). The investigators speculate that the differences between the English and Chinese written languages may account for the different brain regions involved. Reading English requires putting together letters that represent sounds. Reading Chinese, in contrast, requires putting together symbols that represent meanings. Indeed, reading Chinese requires mastery of nearly 6000 different symbols. Thus, Chinese relies more on visual processing, while English relies more on sound processing.

### Aetiology of dyscalculia

There is evidence of some genetic influence on individual variations in maths skills. In particular, the type of maths disability that involves poor semantic memory is most likely to be heritable. Furthermore, the evidence suggests that any genes associated with dyscalculia are also associated with mathematics ability (Plomin & Kovas, 2005).

fMRI studies of people with dyscalculia suggest that areas of the parietal lobe are less active during tasks

Interventions for dyslexia have improved children's reading.



requiring mathematics. Specifically, an area called the intraparietal sulcus has been implicated in dyscalculia (Wilson & Dehaene, 2007).

Researchers have investigated whether dyscalculia might be linked to dyslexia in terms of the cognitive deficits that are associated with both of these types of specific learning disorders. That is, children who have problems with phonological awareness might have problems not only with reading but also with the symbols and numbers in mathematics. The evidence suggests, however, that these two learning disorders are somewhat independent (Jordan, 2007). Children with both dyslexia and dyscalculia have deficits in phonological awareness, but children with only dyscalculia do not. Children with dyscalculia have trouble with tasks requiring manipulation of numbers, whether with actual numbers or with the use of calculations, as in estimating size, but children with dyslexia do not (Landerl et al., 2009).

## Treatment of specific learning disorder

Several strategies are used to treat specific learning disorder, both in school programs and in private tutoring. Traditional linguistic approaches, used primarily in cases of reading and writing difficulties, focus on instruction in listening, speaking, reading and writing skills in a logical, sequential and multi-sensory manner, such as reading out loud under close supervision. In young children, readiness skills, such as letter discrimination, phonetic analysis and learning letter–sound correspondences, may need to be taught before explicit instruction in reading is attempted. Phonics instruction involves helping children master the task of converting sounds to words. Findings from the National Reading Panel, a comprehensive review of the research on teaching children to read, indicate that phonics instruction is beneficial for children with reading difficulties (National Institute of Child Health and Human Development, 2000). As in the clinical case of Marcus described earlier, people with dyslexia can often succeed in college with the aid of instructional supports, such as podcast or webcast lectures that can be re-reviewed, tutors and untimed tests. Colleges are required by law to provide special services to help such students, and public schools are now required to provide transitional vocational and career planning for older adolescents with learning disorders.

Despite early promise in uncontrolled trials (Tallal et al., 1996) a commercially available computer-based learning treatment called Fast ForWord does not appear to be effective in improving speech, language or auditory processing skills. A meta-analysis of six randomised controlled trials found that Fast ForWord was not effective (Strong, Torgerson, Torgerson, & Hulme, 2011).

## 10.5 Intellectual disability

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**LEARNING OUTCOME 10.5** Discuss the description and diagnosis of intellectual disability and the current research on causes and treatments.

In DSM-IV-TR, mental retardation was the name of the disorder that was renamed **intellectual disability** in DSM-5. Why the change? After all, many people are familiar with the term *mental retardation*. However, this is not the term that most mental health professionals use or prefer, perhaps due to the stigma associated with this older term. Most mental health professionals follow the guidelines of the American Association on Intellectual and Developmental Disabilities (AAIDD) or, in our region, the Australasian Society for Intellectual Disability (ASID).

The AAIDD is an organisation whose mission is to ‘promote progressive policies, sound research, effective practices, and universal human rights for people with intellectual and developmental disabilities’ (www.aaid.org). This group changed its name in 2006 (it was formerly known as the American Association of Mental Retardation) in large part to acknowledge that *intellectual disability* is now the preferred term over *mental retardation* (Schalock, Luckasson, & Shogren, 2007). The current AAIDD guidelines are summarised in table 10.4.

**TABLE 10.4** The AAIDD definition of intellectual disability

Intellectual disability is characterised by significant limitations both in intellectual functioning and in adaptive behaviour as expressed in conceptual, social and practical adaptive skills.

This disability begins before age 18.

*Five Assumptions Essential to the Application of the Definition*

1. Limitations in present functioning must be considered within the context of community environments typical of the individual's age, peers, and culture.
2. Valid assessment considers cultural and linguistic diversity as well as differences in communication, sensory, motor and behavioral factors.
3. Within an individual, limitations often coexist with strengths.
4. An important purpose of describing limitations is to develop a profile of needed supports.
5. With appropriate personalised supports over a sustained period, the life functioning of the person with intellectual disability generally will improve.

**Source:** © 2002 American Association on Intellectual and Developmental Disabilities, from [http://aaidd.org/publications/bookstore-home/product-listing/2013/06/24/intellectual-disability-definition-classification-and-systems-of-supports-\(11th-edition\)#.U-KIA4BdXsQ](http://aaidd.org/publications/bookstore-home/product-listing/2013/06/24/intellectual-disability-definition-classification-and-systems-of-supports-(11th-edition)#.U-KIA4BdXsQ)

## Diagnosis and assessment of intellectual disability

The DSM-5 diagnostic criteria for intellectual disability include three criteria: (1) deficits in intellectual functioning, (2) deficits in adaptive functioning, and (3) an onset during development (i.e., as a child).

### DSM-5

#### DSM-5 criteria for intellectual disability

- People with an intellectual disability have intellectual deficits (e.g., in solving problems, reasoning, abstract thinking) determined by intelligence testing and broader clinical assessment.
- People with intellectual disability experience significant deficits in adaptive functioning relative to their age and cultural group in one or more of the following areas: communication, social participation, work or school, independence at home or in the community, requiring the need for support at school, work or independent life.
- The onset of these deficits occurs during child development.

The first two criteria make the DSM-5 consistent with the approach of the AAIDD. First, there is explicit recognition that an IQ score must be considered within the context of a more thorough assessment. Second, adaptive functioning must be assessed across a broad range of domains. Finally, the DSM-5 no longer distinguishes among mild, moderate and severe intellectual disability based on IQ scores alone, as was done in DSM-IV-TR. The severity of intellectual disability is assessed in three domains: conceptual (which includes intellectual and other cognitive functioning), social and practical.

The AAIDD approach encourages the identification of an individual's strengths and weaknesses on psychological, physical and environmental dimensions with a view towards determining the kinds and degrees of support needed to enhance the person's functioning in different domains. Consider Roger, a 24-year-old man with an IQ of 45 who has attended a special program for people with intellectual disability since he was six. The AAIDD approach would emphasise what is needed to maximise Roger's functioning. Thus, a clinician might discover that Roger can use the bus system if he takes a route familiar to him, and thus he might be able to go to a movie by himself from time to time. And although he cannot prepare complicated meals, he might be able to learn to prepare frozen dinners in a microwave oven. The assumption is that by building on what he can do, Roger will make more progress. We hope that the DSM-5 approach will also work like this.

In the schools, an individualised educational program (IEP) is based on the person's strengths and weaknesses, and on the amount of instruction needed. Students are identified by the classroom environment they are judged to need. This approach can lessen the stigmatising effects of having intellectual disability and may also encourage a focus on what can be done to improve the student's learning.

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When assessing adaptive behaviour, the cultural environment must be considered. A person living in a rural community may not need the same skills as those needed by someone living in Sydney, and vice versa.



## Aetiology of intellectual disability

At this time, the primary cause of intellectual disability can be identified in only 25 percent of the people affected. The causes that can be identified are typically neurobiological.

### Genetic or chromosomal abnormalities

One chromosomal abnormality that has been linked with intellectual disability is as **Down Syndrome (trisomy 21)**, which refers to having an extra copy (i.e., three instead of two) of chromosome 21.

People with Down syndrome may have intellectual disability as well as some distinctive physical signs, such as short and stocky stature; oval, upward-slanting eyes; a prolongation of the fold of the upper eyelid over the inner corner of the eye; sparse, fine, straight hair; a wide and flat nasal bridge; square-shaped ears; a large, furrowed tongue, which may protrude because the mouth is small and its roof low; and short, broad hands.

Another chromosomal abnormality that can cause intellectual disability is **fragile X syndrome**, which involves a mutation in the *fMRI* gene on the X chromosome (National Fragile X Foundation, n.d.). Physical symptoms associated with fragile X include large, underdeveloped ears and a long, thin face. Many people with fragile X syndrome have intellectual disability. Others may not have intellectual disability but may have a specific learning disorder, difficulties on neuropsychological tests and mood swings. About a third of children with fragile X syndrome also exhibit autism spectrum behaviours, suggesting that the *fMRI* gene may be one of the many genes that contribute to autism (Hagerman, 2006).



### Recessive-gene diseases

Several hundred recessive-gene diseases have been identified, and many of them can cause intellectual disability. Here we discuss one recessive-gene disease, phenylketonuria.

In **phenylketonuria (PKU)**, the infant, born without obvious signs of difficulty, soon begins to suffer from a deficiency of a liver enzyme, phenylalanine hydroxylase. Because of this enzyme deficiency, phenylalanine and its derivative, phenylpyruvic acid, are not broken down and instead build up in the body's fluids. This build-up can damage the brain because the unmetabolised amino acid interferes with the process of myelination, the sheathing of neuron axons, which is essential for neuronal function. Myelination supports the rapid transmittal of neuronal impulses. If not properly treated, intellectual disabilities can be profound.

Although PKU is rare, with an incidence of about 1 in 15 000 live births, it is estimated that 1 person in 70 is a carrier of the recessive gene. A blood test is available for prospective parents who have reason to suspect that they might be carriers. Pregnant women who carry the recessive gene must monitor their diet closely so that the foetus will not be exposed to toxic levels of phenylalanine. State laws require testing newborns for PKU. If the test is positive, the parents are taught to provide the infant a diet low in phenylalanine.

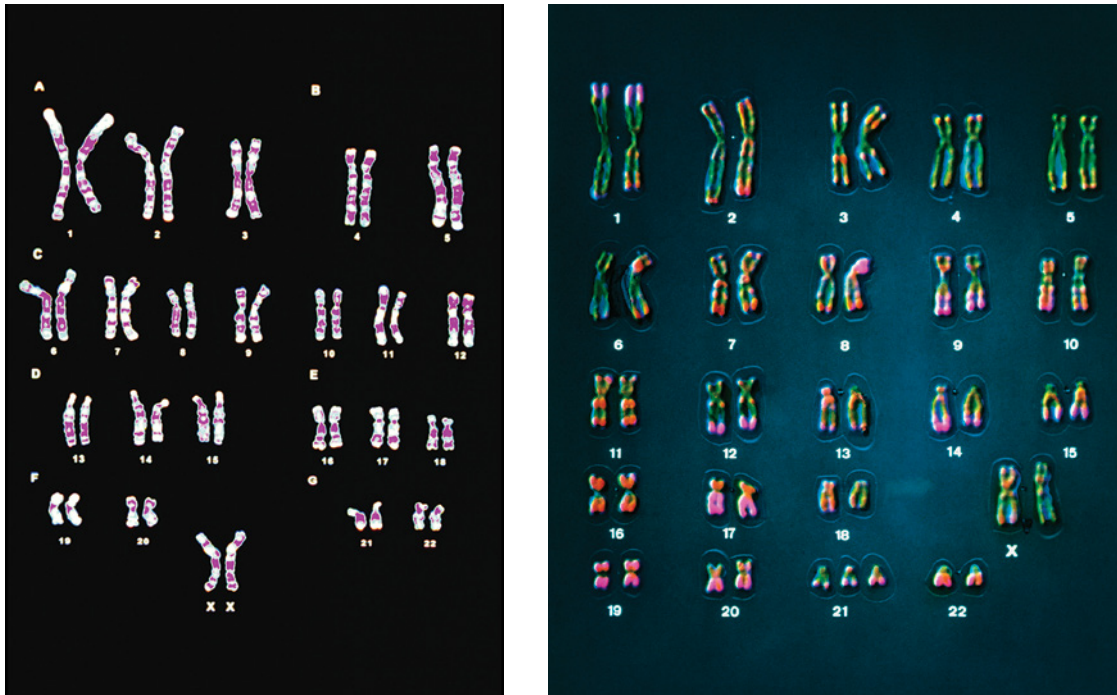
Parents are encouraged to introduce the special diet as early as possible and to maintain it indefinitely. Studies have indicated that children whose dietary restrictions stop between the ages of five and seven begin to show subtle declines in functioning, particularly in IQ, reading and spelling (Fishler, Azen, Henderson, Friedman, & Koch, 1987). Even among children with PKU who maintain the diet, however, deficits in perceptual, memory and attentional abilities have been observed (Banich, Passarotti, White, Nortz, & Steiner, 2000; Huijbregts et al., 2002).

### Infectious diseases

While in utero the foetus is at increased risk of intellectual disabilities resulting from maternal infectious diseases, such as rubella, cytomegalovirus, toxoplasmosis, herpes simplex and HIV. The consequences

of these diseases are most serious during the first trimester of pregnancy, when the foetus has no detectable immunological response, that is, its immune system is not developed enough to ward off infection. The mother may experience slight or no symptoms from the infection, but the effects on the developing foetus can be devastating.

The normal complement of chromosomes is 23 pairs (figure on the left). In Down syndrome, there are three copies (a trisomy) of chromosome 21 (figure on the right).



Infectious diseases can also affect a child's developing brain after birth. Encephalitis and meningococcal meningitis may cause brain damage and even death if contracted in infancy or early childhood. In adulthood, these infections are usually far less serious. There are several forms of childhood meningitis, a disease in which the protective membranes of the brain are acutely inflamed and fever is very high.

### Environmental hazards

Several environmental pollutants are implicated in intellectual disability. One such pollutant is mercury, which may be ingested by eating affected fish. Another is lead, which is found in lead-based paints, smog and the exhaust from automobiles that burn leaded gasoline. Lead poisoning can cause kidney and brain damage as well as anaemia, intellectual disabilities, seizures and death. Lead-based paint is now prohibited in the United States, but it is still found in older homes, where children may eat pieces that flake off.

## Treatment of intellectual disability

### Residential treatment

Since the 1960s, serious and systematic attempts have been made to educate children with intellectual disability as fully as possible. Although many people can acquire the competence needed to function effectively in the community, some people need the extra support of a residential treatment program.

Ideally, adults with intellectual disability in need of such support live in small to medium-sized residences that are integrated into the community. Medical care is provided, and trained, live-in supervisors and aides help with residents' special needs around the clock. Residents are encouraged to participate

in household routines to the best of their abilities. Many adults with intellectual disability have jobs and are able to live independently in their own apartments. Others live semi-independently in apartments housing three to four adults; generally, a counsellor provides aid in the evening.

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Newborn babies in Australia and New Zealand are routinely screened for PKU. If excess phenylalanine is found in the blood, a special diet is recommended for the baby.



### Behavioural treatments

Early-intervention programs using behavioural techniques have been developed to improve the level of functioning of people with intellectual disability. Specific behavioural objectives are defined, and children are taught skills in small, sequential steps (Reid, Wilson, & Faw, 1991).

To teach a child a particular routine, the therapist usually begins by dividing the targeted behaviour, such as eating, into smaller components: pick up spoon, scoop food from plate onto spoon, bring spoon to mouth, remove food with lips, chew and swallow food. Operant conditioning principles are then applied to teach the child these components of eating. For example, the child may be reinforced for successive approximations to picking up the spoon until he or she is able to do so. This operant approach, sometimes called *applied behaviour analysis*, is also used to reduce inappropriate and self-injurious behaviour. Reinforcing substitute behaviours can often reduce these behaviours.

### Cognitive treatments

Many children with intellectual disability have difficulty using strategies in solving problems, and when they do use

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Although lead-based paint is now illegal, it can still be found in older homes. Eating these paint chips can cause lead poisoning, which can cause intellectual disability.



strategies, they often do not use them effectively. Self-instructional training teaches these children to guide their problem-solving efforts through speech.

For example, one group of researchers taught high school students with intellectual disability to make their own buttered toast and clean up after themselves (Hughes, Hugo, & Blatt, 1996). A teacher would demonstrate and verbalise the steps involved in solving a problem, such as the toaster being upside down or unplugged. The young people learned to talk themselves through the steps using simple verbal or signed instructions. For example, when the toaster was presented upside down, the person would be taught to first state the problem ('Won't go in'), then to state the response ('Turn it'), self-evaluate ('Fixed it'), and self-reinforce ('Good'). They were rewarded with praise and high-fives when they verbalised and solved the problem correctly. Several studies have demonstrated that even people with severe intellectual disability can learn self-instructional approaches to problem solving and then generalise the strategy to new tasks, including taking lunch orders at a cafeteria and performing janitorial duties (Hughes & Agran, 1993).

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Computer-assisted instruction is well suited for applications in the treatment of intellectual disability.



### Computer-assisted instruction

Computer-assisted instruction is increasingly found in educational and treatment settings of all kinds; it may be especially well suited to the education of people with intellectual disability. The visual and auditory components of computers can help to maintain the attention of distractible students; the level of the material can be geared to the individual, ensuring successful experiences; and the computer can meet the need for numerous repetitions of material without becoming bored or impatient, as a human teacher might. For example, computers have been used to help people with intellectual disability learn to use an ATM (Davies, Stock, & Wehmeyer, 2003). Smartphones can be enormously helpful by serving as aids for reminders, directions, instructions and daily tasks.

## 10.6 Autism spectrum disorder

**LEARNING OUTCOME 10.6** Describe the symptoms, causes and treatments for autism spectrum disorders.

Although autism was first described about 70 years ago (see focus on discovery 10.3 for the history of autism), it was not formally included in the DSM until the third edition, published in 1980. As discussed in focus on discovery 10.2, the rates of autism spectrum disorder have been rising over the past 20 years. With this increase in prevalence has come an increase in research on the causes of this disorder.

### Clinical descriptions, prevalence and prognosis of autism spectrum disorder

In DSM-5, four diagnostic categories from DSM-IV-TR — autistic disorder, Asperger's disorder, pervasive developmental disorder not otherwise specified, and childhood disintegrative disorder — were combined into one category called **autism spectrum disorder**. Why the change? Research conducted on the different DSM-IV-TR categories did not support the distinct categories. In other words, these disorders all share similar clinical features and aetiologies and seem to vary only in severity. Thus, DSM-5 has one disorder category, autism spectrum disorder (ASD), which includes different clinical specifiers relating to severity and the extent of language impairment.

The DSM-5 criteria for ASD are presented in the box below. In the next sections, we describe the clinical features, focusing on problems in social and emotional interactions and in communication as well as on repetitive or ritualistic behaviours.

#### Social and emotional disturbances

Children with ASD can have profound problems with the social world (Dawson et al., 2004). They may rarely approach others and may look through or past people or turn their backs on them. For example, one study found that children with ASD rarely offered a spontaneous greeting or farewell (either verbally or through smiling, making eye contact, or gesturing) when meeting or departing from an adult (Hobson & Lee, 1998). Few children with ASD initiate play with other children, and they are usually unresponsive to anyone who approaches them.

#### FOCUS ON DISCOVERY 10.3

##### A brief history of autism spectrum disorder

Autism was identified in 1943 by a psychiatrist at Johns Hopkins, Leo Kanner, who, in the course of his clinical work, noted 11 disturbed children who behaved in ways that were not common in children with intellectual disability or schizophrenia. He named the syndrome *early infantile autism* because he observed that 'there is from the start an extreme autistic aloneness that, whenever possible, disregards, ignores, shuts out anything that comes to the child from the outside' (Kanner, 1943).

Kanner considered autistic aloneness to be the most fundamental symptom. He also learned that these 11 children had been unable from the beginning of life to relate to people in the ordinary way. They were severely limited in language and had a strong, obsessive desire for everything about them to remain unchanged. Despite its early description by Kanner and others (Rimland, 1964), the disorder was not accepted into official diagnostic nomenclature until the publication of DSM-III in 1980, where it was called autistic disorder.

Asperger's disorder was named after Hans Asperger, who in 1944 described the syndrome as being less severe and having fewer communication deficits than autism. It was first introduced to the DSM in 1994 in DSM-IV. Social relationships are poor and stereotyped behaviour is intense and rigid, but language and intelligence are intact. Because research suggests that Asperger's disorder does not differ qualitatively from autistic disorder, these two categories will likely be combined in DSM-5. Nevertheless, more research has been conducted in the last 10 years on Asperger's disorder, perhaps due to the recognition of this condition among adults who for years wondered why they were different from others. Adults with the DSM-IV-TR diagnosis Asperger's disorder are now more frequently recognised and treated by mental health professionals (Gaus, 2007).

People formerly in the Asperger's disorder category but now under the broader domain of ASD will likely continue to seek and receive the support and help they need. Researchers, clinicians and families have referred to the 'autism spectrum' for years, so, in some ways, this change might not be so difficult to accommodate, at least in terms of the name. However, some worry that the stigma associated with the name *autism* might keep people from seeking help (see focus on discovery 2.3 on the underreporting of stigmatised behaviour). On the other hand, some areas, such as the states of California and Texas in the US, mandate services for children with autism but not Asperger's, and thus more people might get help.

### QUESTIONS

1. Why do you think Asperger's disorder has gone under a name change in the latest DSM-5 to become autism spectrum disorder?
2. What are some common characteristics or symptoms of ASD?

Children with ASD sometimes make eye contact, but their gaze may have an unusual quality. Typically, children gaze to gain someone's attention or to direct the other person's attention to an object; children with ASD generally do not (Dawson et al., 2004). This is often referred to as a problem in **joint attention**. That is, interactions that require two people to pay attention to each other, whether speaking or communicating emotion nonverbally, are impaired in children with autism.

More fine-grained analyses of gaze involve measuring eye movements and looking time. In one study, six-month old infants later diagnosed with ASD spent less time looking at videos of dynamic speaking faces, particularly in the eyes and mouth regions, compared to typically developing children (Shic, Macari, & Chawarska, 2014). A different study measured the eye movements of infants several different times between the ages of 2 months and 24 months (Jones & Klin, 2013). At two months, the eye movements of infants who later were diagnosed with ASD did not differ from typically developing infants, suggesting that children with ASD are not born with gaze deficits. However, between two months and 24 months, the pattern of gaze between the two groups of infants began to diverge. Overall, the time spent looking at faces declined in the group of infants with ASD, so much so that by two years of age, these children were looking at the faces 50 percent less than children without ASD. Infants who showed a faster decline in looking time, particularly in the eyes region, were the ones who later had more social deficits.

Waleed Aly, a co-host of the Australian television show, *The Project* — pictured with wife, Susan Carland — has publicly opened up about his son's autism.



Consistent with the findings showing that children with ASD spend less time looking at other people's faces, fMRI studies have found that people with ASD do not show activation in the fusiform gyrus, other regions in the temporal lobes, and the amygdala, the areas of the brain most often associated with identifying faces and emotion, when completing face perception or identity tasks (Critchley et al., 2001; Pierce, Haist, Sedaghat, & Courchesne, 2004; Pierce, Muller, Ambrose, Allen, & Courchesne, 2001). Instead, other areas of the brain show activation during these tasks, suggesting perhaps a less efficient system for identifying faces.

Some researchers have proposed that children with ASD have a deficient 'theory of mind' that is at the core of the kinds of social dysfunctions we have described here (Gopnik, Capps, & Meltzoff, 2000; Sigman, 1994). *Theory of mind* refers to a person's understanding that other people have desires, beliefs, intentions and emotions that may be different from one's own. This ability is crucial for understanding and successfully engaging in social interactions. Theory of mind typically develops between two and a half and five years of age. Children with ASD seem not to undergo this developmental milestone and thus seem unable to understand others' perspectives and emotional reactions.

#### DSM-5

##### DSM-5 criteria for autism spectrum disorder

- (a) Deficits in social communication and social interactions as exhibited by the following:
  - deficits in social or emotional reciprocity such as not approaching others, not having a back-and-forth conversation, reduced sharing of interests and emotions
  - deficits in nonverbal behaviours such as eye contact, facial expression, body language
  - deficit in development of peer relationships appropriate to developmental level.
- (b) Restricted, repetitive behaviour patterns, interests, or activities exhibited by at least two of the following:
  - stereotyped or repetitive speech, motor movements, or use of objects
  - excessive adherence to routines, rituals in verbal or nonverbal behaviour, or extreme resistance to change
  - very restricted interests that are abnormal in focus, such as preoccupation with parts of objects
  - hyper- or hyporeactivity to sensory input or unusual interest in sensory environment, such as fascination with lights or spinning objects.
- (c) Onset of autism spectrum disorder occurs during early childhood.
- (d) The symptoms of autism spectrum disorder limit and impair functioning.

Although some children with ASD can learn to understand emotional experiences, they 'answer questions about ... emotional experiences like normal children answer difficult arithmetic questions' (Sigman, 1994, p. 151), with concentrated effort. Laboratory studies of children with ASD have found that they may recognise others' emotions without really understanding them (Capps, Rasco, Losh & Heerey, 1999; Capps, Yirmiya, & Sigman, 1992). For example, when asked to explain why someone was angry, a child with ASD responded 'because he was yelling' (Capps, Losh, & Thurber, 2000).

### Communication deficits

Even before they acquire language, some children with ASD show deficits in communication. Babbling, a term describing the utterances of infants before they begin to use words, is less frequent in infants with ASD and conveys less information than it does in other infants (Ricks, 1972). By two years of age, most typically developing children use words to represent objects in their surroundings and construct one- and two-word sentences to express more complex thoughts, such as 'Mummy go' or 'Me juice'. In contrast, children with ASD lag well behind in these abilities and often show other language disturbances.

One such feature associated with ASD is echolalia, in which the child echoes, usually with remarkable fidelity, what he or she has heard another person say. The teacher may ask a child with ASD, 'Do you want a cookie?' The child's response may be, 'Do you want a cookie?' This is immediate echolalia. In delayed

echolalia, the child may be in a room with the television on and appear to be completely uninterested. Several hours later or even the next day, the child may echo a word or phrase from the television program.

Another language abnormality common in the speech of children with ASD is **pronoun reversal**, in which children refer to themselves as 'he,' 'she,' or 'you' (or even by their own name). For example:

**Parent:** What are you doing, Johnny?

**Child:** He's here.

**Parent:** Are you having a good time?

**Child:** He knows it.

Pronoun reversal is closely linked to echolalia — when children with ASD use echolalic speech, they refer to themselves as they have heard others speak of them and misapply pronouns. Children with ASD are very literal in their use of words. If a father provided positive reinforcement by putting his daughter on his shoulders when she learned to say the word *yes*, then the child might say *yes* to mean she wants to be lifted onto her father's shoulders. Or a child may say 'do not drop the cat' to mean 'no', because a parent had used these emphatic words when the child was about to drop the family feline.

### **Repetitive and ritualistic acts**

Children with ASD can become extremely upset over changes in their daily routines and surroundings. An offer of milk in a different drinking cup or a rearrangement of furniture may make them cry or may precipitate a temper tantrum.

An obsessional quality may pervade the behaviour of children with ASD. In their play, they may continually line up toys or construct intricate patterns with household objects. As they grow older, they may become preoccupied with train schedules, subway routes and number sequences. Children with ASD are also likely to perform a more limited number of behaviours than children without ASD and are less likely to explore new surroundings.

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Children with ASD do not often play or socially interact with other children.



Children with ASD may also display stereotypical behaviour, peculiar ritualistic hand movements and other rhythmic movements, such as endless body rocking, hand flapping and walking on tiptoe. They may spin and twirl string, crayons, sticks, and plates, twiddle their fingers in front of their eyes, and stare at fans and other spinning things. Researchers often describe these as self-stimulatory activities. The children may become preoccupied with manipulating an object and may become very upset when interrupted.

Some children with ASD can become preoccupied with and form strong attachments to simple inanimate objects (e.g., keys, rocks, a wire-mesh basket, light switches, a large blanket) and to more complex mechanical objects (e.g., refrigerators and vacuum cleaners). If the object is something they can carry, they may walk around with it in their hands, and this may interfere with their learning to do more useful things.

## Comorbidity and ASD

Many children with ASD also have intellectual disability. However, sensorimotor development is the area of greatest relative strength among these children. These children, who may show severe and profound deficits in cognitive abilities, can be quite graceful and adept at swinging, climbing or balancing, whereas children with intellectual disability have far more difficulty in areas of gross motor development, such as learning to walk. Sometimes children with ASD may have isolated skills that reflect great talent, such as the ability to multiply two four-digit numbers rapidly in their heads. They may also have exceptional long-term memory, being able to recall the exact words of a song heard years earlier.

One study found that over a third of the children with ASD also have a specific learning disorder (Lichtenstein, Carlstrom, Ramstam, Gillberg, & Anckarsater, 2010). In addition, ASD is also frequently comorbid with anxiety, including separation anxiety, social anxiety, general anxiety and specific phobias (White, Oswald, Ollendick, & Scahill, 2009).

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People with ASD may engage in stereotyped behaviour, such as ritualistic hand movements.



## Prevalence of autism spectrum disorder

ASD begins in early childhood and can be evident in the first months of life. It affects about 1 of every 68 children (CDC, 2014). About five times more boys than girls have ASD (CDC, 2014). There has been a large increase in the number of ASD diagnoses over the past 25 years (see focus on discovery 10.2 for more on this). It is found in all socioeconomic, ethnic and racial groups. The diagnosis of ASD is remarkably stable. In one study, only 1 out of 84 children diagnosed with ASD at age 2 no longer met the diagnostic criteria at age 9 (Lord et al., 2006).

## Prognosis for autism spectrum disorder

What happens to children with ASD when they reach adulthood? Generally, children with higher IQs who learn to speak before age six have the best outcomes. For example, one longitudinal study of children with ASD from preschool to early adulthood found that IQs over 70 predicted more strengths and fewer weaknesses in adaptive functioning as they grew older (McGovern & Sigman, 2005), and outcomes were better for those who had interacted and engaged more with their peers. Other studies of people with ASD who have higher IQ scores have indicated that most do not require residential care and some are able to attend college and support themselves through employment (Yirmiya & Sigman, 1991). Still, many independently functioning adults with ASD continue to show impairment in social relationships (Howlin, Goode, Hutton, & Rutter, 2004; Howlin, Mawhood, & Rutter, 2000). Focus on discovery 10.4 describes a woman with ASD whose adult life is remarkable for its professional distinction blended with the social and emotional deficits that are part of ASD.

## Aetiology of autism spectrum disorder

The earliest theory about the aetiology of ASD was that psychological factors such as bad parenting were responsible for its development. This narrow and faulty perspective has been replaced by theories based on evidence that genetic and neurological factors are important in the aetiology. Despite the lack of empirical support for early psychological theories, they gained enough recognition to place a tremendous emotional burden on parents who were told that they were at fault for their child's ASD.

### Genetic factors

Evidence suggests a genetic component for ASD, with heritability estimates of around .80 (Lichtenstein et al., 2010; Sullivan et al., 2012). The risk of ASD or language delay among siblings of people with the disorder is much higher than it is among siblings of people who do not have ASD (Constantino, Zhang, Frazier, Abbachi, & Law, 2010). Evidence for genetic transmission of ASD comes from twin studies, which have found between 47 and 90 percent concordance for ASD between identical twins, compared with concordance rates of 0 to 20 percent between fraternal twins (Bailey et al., 1995; Le Couteur et al., 1996; Lichtenstein et al., 2010).

Even though genes play a big role in ASD, remember that genes do their work via the environment. Another twin study used the most current and well-validated method for diagnosing ASD rather than relying solely on medical records or parental reports as other studies have done. This study found that shared environmental factors (e.g., common experiences in a family; see chapter 1) accounted for over half of the risk for developed autism (Hallmayer et al., 2011).

Studies of twins and families with a member with ASD suggest that ASD is linked genetically to a broader spectrum of deficits in communication and social interaction. In families where more than one child had ASD or language delay, unaffected siblings also exhibited deficits in social communication and interactions (Constantino et al., 2010).

Molecular genetics studies try to pinpoint areas of the genome that may confer risk for ASD. Recall from chapter 1 that genome-wide association studies (GWAS) look for differences in gene sequence (single nucleotide polymorphisms — SNPs) and gene structure (copy number variations — CNVs).

One research group studying CNVs found that a deletion on chromosome 16 was associated with ASD in three different samples (Weiss, Shen, Korn et al., 2008). The deletion represents a genetic flaw — it was not supposed to be deleted — and the researchers suggest that, although it is not clear why the flaw occurs, it is nonetheless associated with an increased risk of developing ASD. Other GWAS studies have identified SNPs between two genes on chromosome 5 that have been replicated in two independent samples of people with ASD (Wang et al., 2009) and one sample of people without ASD but who had communication and social-emotional difficulties (St. Pourcain et al., 2010).

### Neurobiological factors

More and more research is linking the language, social and emotional deficits in ASD to the brain. A number of studies examining the brain in ASD have been well replicated, allowing for a clearer picture of what may go wrong in the brain among people with ASD. What remains to be figured out is why the brain goes awry early in development.

Studies using magnetic resonance imaging (MRI) found that, overall, the brains of adults and children with ASD are larger than the brains of adults and children without ASD (Courchesne, Carnes, & Davis, 2001; Piven et al., 1995; Piven, Arndt, Bailey, & Andreasen, 1996). This same finding has been supported by studies using the measurement of head circumference as an indicator of brain size (Courchesne, Carper, & Akshoomoff, 2003), but studies using this measure don't always find support for larger brain size in ASD (Raznahan et al., 2013). What makes these findings more interesting and puzzling is that most children with ASD are born with brains of a relatively normal size; however, between the ages of two and four, the brains of children with ASD become significantly larger (Courchesne, 2004). One longitudinal study assessed brain size using MRI when children with and without autism were two years old and again when they were four or five years old. The researchers found that the children with autism had larger brain size at age two but that it did not continue to increase at ages four or five, thus suggesting that brain growth does not continue past the first few years of life (Hazlett et al., 2011).

Having a larger-than-normal brain is not necessarily a good thing, as it might indicate that neurons are not being pruned correctly. The pruning of neurons is an important part of brain maturation; older children have fewer connections between neurons than do babies. Adding further to this puzzle, brain growth in ASD appears to slow abnormally in later childhood. We do not yet know how this pattern of brain growth is linked to the signs and symptoms of ASD. It is worth noting that the areas of the brain that are 'overgrown' in ASD include the frontal, temporal and cerebellar, which have been linked with language, social and emotional functions.

#### FOCUS ON DISCOVERY 10.4

##### The story of a woman living with autism spectrum disorder

Temple Grandin is a woman with autism spectrum disorder. She also has a PhD in animal science, runs her own business designing machinery for use with farm animals, and is on the faculty at Colorado State University. Four autobiographical books (Grandin 1986, 1995, 2008, 2013) and a profile by neurologist Oliver Sacks (1995) provide a moving and revealing portrait of the mysteries of ASD. A highly acclaimed and awarded HBO movie based on Grandin's 1995 book *Thinking in Pictures* was released in 2010 and starred Clare Danes as Temple. Grandin has also written other books about her professional work with animals.

Lacking understanding of the complexities and subtleties of human social discourse, and deficient in the ability to empathise with others, Grandin sums up her relationship to the non-autistic world by saying, 'Much of the time I feel like an anthropologist on Mars' (Sacks, 1995, p. 259).

Grandin was diagnosed with autism in 1950 at age three. She had no speech at all, and doctors predicted that institutionalisation would be her fate. However, with the help of a therapeutic nursery school, speech therapy, and the support of her family, she learned to speak by age six and began to make more contact with others. Still, as an adolescent observing other children interact, Grandin 'sometimes wondered if they were all telepathic' (Sacks, 1995, p. 272), so mysterious did she find the ability of normal children to understand each other's needs and wishes, to empathise, to communicate. In her own writings, Grandin

points out that many people with ASD are great fans of *Star Trek*, especially the characters of Spock and Data, the former a member of the Vulcan race, purely intellectual, logical beings who eschew any consideration of the emotional side of life, and the latter an android, a highly sophisticated computer housed in a human body and, like Spock, lacking in emotion. (One of the dramatic themes involving both characters was, of course, their flirtation with the experience of human emotion, portrayed with particular poignancy by Data. This is a theme in Grandin's life as well.) As Grandin (1995) wrote at age 47:

All my life I have been an observer, and I have always felt like someone who watches from the outside. I could not participate in the social interactions of high school life.

Even today, personal relationships are something I don't really understand. I've remained celibate because doing so helps me avoid the many complicated situations that are too difficult for me to handle. [M]en who want to date often don't understand how to relate to a woman. They [and I myself] remind me of Data, the android on *Star Trek*. In one episode, Data's attempts at dating were a disaster. When he tried to be romantic [by effecting a change in a subroutine of his computer program], he complimented his date by using scientific terminology. Even very able adults with autism have such problems. (pp. 132–133)

Some of the difficulties of people with ASD make them charmingly honest and trustworthy. 'Lying,' wrote Grandin, 'is very anxiety-provoking because it requires rapid interpretations of subtle social cues [of which I am incapable] to determine whether the other person is really being deceived' (Grandin, 1995, p. 135).

Grandin's professional career is impressive. She uses her remarkable powers of visualisation and her empathy for farm animals to design machines such as a chute leading cows to slaughter that takes them on a circular route, protecting them from awareness of their fate until the moment of death. She has also designed and built a 'squeeze machine', a device that provides comforting hugs without the need for human contact. It has 'two heavy, slanting wooden sides, perhaps four by three feet each, pleasantly upholstered with a thick, soft padding. They [are] joined by hinges to a long, narrow bottom board to create a V-shaped, body-sized trough. There [is] a complex control box at one end, with heavy-duty tubes leading off to another device, in a closet. [An] industrial compressor exerts a firm but comfortable pressure on the body, from the shoulders to the knees' (Sacks, 1995, pp. 262–263). Her explanation of the rationale behind this contraption is that as a little girl she longed to be hugged but was also very fearful of physical contact with another person. When a favourite, large-bodied aunt hugged her, she felt both overwhelmed and comforted. Terror commingled with pleasure.

She started to have daydreams — she was just five at the time — of a magic machine that could squeeze her powerfully but gently, in a huglike way, and in a way entirely commanded and controlled by her. Years later, as an adolescent, she had seen a picture of a squeeze chute designed to hold or restrain calves and realized that that was it: a little modification to make it suitable for human use, and it could be her magic machine. (Sacks, 1995, p. 263)

After watching her demonstrate the machine and trying it himself, Sacks observed:

It is not just pleasure or relaxation that Temple gets from the machine but, she maintains, a feeling for others. As she lies in her machine, she says, her thoughts often turn to her mother, her favorite aunt, her teachers. She feels their love for her, and hers for them. She feels that the machine opens a door into an otherwise closed emotional world and allows her, almost teaches her, to feel empathy for others. (Sacks, 1995, p. 264)

Accounts such as those of Grandin and Sacks can provide insight into how people adapt to their own idiosyncrasies, using the sometimes peculiar gifts they have been given and working around the difficulties they experience. 'Autism, while it may be pathologised as a syndrome, must also be seen as a whole mode of being, a deeply different mode or identity, one that needs to be conscious (and proud) of itself,' wrote Sacks (1995, p. 277). 'At a public lecture, Temple ended by saying, "If I could snap my fingers and be non-autistic, I would not — because then I wouldn't be me. Autism is part of who I am"' (Sacks, 1995, p. 291).

This type of sentiment was emphasised in Grandin's most recent book (2013) where she stated that there are many things *right* with the autistic brain. In other words, some of the difficulties experienced by people with autism can actually be strengths. For example, one of the reasons that social interactions are difficult for her to decipher is that this requires an integration of many things happening all at once (e.g., words, facial expressions, gestures, tone of voice). However, she is quite good at focusing on one thing at a time, and this means she is really good at paying attention to small details. She believes that this is why she has been so successful with animals; she is able to see a small detail that might scare an animal that other people would ignore.

Temple Grandin, PhD, was diagnosed with autism in early childhood but has had a successful academic career.



#### QUESTIONS

1. How might those with ASD benefit from learning about Temple Grandin?
2. What do you think Grandin has done for the ASD community?

Other areas of the brain are implicated in ASD as well. A meta-analysis of 46 studies found a large effect size for enlarged cerebellum (Stanfield et al., 2008). Another study found that the commonly observed tendency of children with ASD to explore their surroundings less than other children do is correlated with a larger-than-normal cerebellum (Pierce & Courchesne, 2001).

Given that ASD is associated with social and emotional difficulties, and that the amygdalae are associated with social and emotional behaviour, it stands to reason that the amygdalae might be involved in ASD. One study found that the amygdalae were larger among children with ASD (Munson et al., 2006) and that larger amygdalae at ages three or four predicted more difficulties in social behaviour and communication at age six. This finding is consistent with studies showing overgrowth of other brain areas. However, the other study found that *small* amygdalae size in ASD was correlated with difficulties in emotional face perception and less gaze in the eye region of faces during the perception task (Nacewicz, Dalton, Johnstone et al., 2006). How can we make sense of these seemingly different findings? Participants in the study by Nacewicz and colleagues were older, suggesting that the brain changes that continue throughout in development may be differentially related to social and emotional impairments. A later meta-analysis confirmed that amygdala size was related to age, with older age being associated with smaller amygdala size (Stanfield et al., 2008).

## Treatment of autism spectrum disorder

Even though genetic and neurological factors in the aetiology of ASD have much more empirical support than psychological factors, it is the psychological treatments that currently show the most promise, not medications. The lesson is that a neurological problem may well be treatable psychologically.

Treatments for children with ASD are usually aimed at reducing their unusual behaviour and improving their communication and social skills. In most cases, the earlier the intervention begins, the better the outcome. In a promising longitudinal study, children at high risk for developing ASD (parent or sibling with an ASD) were studied beginning at age 14 months. Even though these children did not yet have language, the researchers were able to identify deficits in joint attention and communication that allowed for an early provisional diagnosis of ASD (Landa, Holman, & Garrett-Mayer, 2007).

### Behavioural treatment

In the late 1980s, Ivar Lovaas conducted an intensive operant conditioning-based program of behavioural treatment with young (under four years old) children with ASD (Lovaas, 1987). Therapy encompassed all aspects of the children's lives for more than 40 hours a week over more than 2 years. Parents were trained extensively so that treatment could continue during almost all the children's waking hours. Nineteen children receiving this intensive treatment were compared with 40 children who received a similar treatment for less than 10 hours per week. Both groups of children were rewarded for being less aggressive, more compliant, and more socially appropriate — for example, talking and playing with other children.

The results of this landmark study were dramatic and encouraging: a larger increase in IQ scores was shown for children in the intensive-therapy group in first grade (after about two years in the intensive therapy) compared to the other group. Furthermore, 9 of the 19 in the intensive-therapy group were promoted to second grade in a regular public school, whereas only one of the much larger group achieved this level of functioning. A follow-up of these children four years later indicated that the intensive-treatment group maintained their gains in IQ, adaptive behaviour and grade promotions in school (McEachin, Smith, & Lovaas, 1993). Although critics rightly pointed out weaknesses in the study's methodology and outcome measures (Schopler, Short, & Mesibov, 1989), this ambitious program demonstrated the benefits of intensive therapy with the heavy involvement of both professionals and parents in dealing with the challenges of ASD.

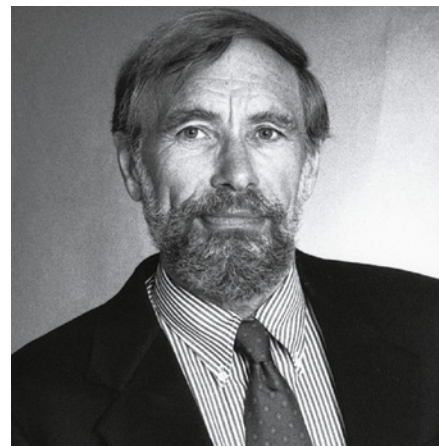
A randomised controlled clinical trial examined the efficacy of intensive behavioural treatment on a broader scale. This study compared an intensive behavioural treatment (about 25 hours a week, instead of 40) to a treatment that consisted of parent training only (Smith, Groen, & Wynn, 2000). Although the behavioural treatment was more effective than parent training alone, the children in this study did not show the same gains as in the study discussed above, perhaps because the treatment was implemented for fewer hours.

A meta-analysis of 22 studies using other types of intensive behavioural treatments, either in a clinic setting or with parents as the primary point of intervention, reported a number of noteworthy results. First, the average quality of these studies, rated on a 1 to 5 scale with 5 being the best, was only 2.5. Few were randomised clinical trials, and many had very small sample sizes. With these limitations in mind, the overall effect sizes were large for changes in IQ, language skills, overall communication, socialisation and daily living skills (Virués-Ortega, 2010). These results are encouraging, but it remains important to conduct more rigorous studies of these types of treatments.

Other interventions seek to improve children's problems in joint attention and communication. In a randomised controlled clinical trial, children ages three and four with ASD were randomly assigned to a joint attention (JA) intervention, a symbolic play (SP) intervention, or a control group (Kasari, Freeman, & Paparella, 2006). All children were already part of an early-intervention program; the JA

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Ivar Lovaas, a behaviour therapist, was noted for his operant conditioning treatment of children with autism.



and SP interventions were additional interventions provided to the children in 30-minute daily blocks for 6 weeks. Children in the JA and SP treatments showed more improvement than children in the control group, and at 6 and 12 months after the treatment, children in the JA and SP groups had greater expressive language skills than children in the control group (Kasari, Paparella, Freeman, & Jahromi, 2008).

### **Drug treatment**

Drug treatment of ASD is, at this point, less effective than behavioural treatment. The most commonly used medication for treating problem behaviours in children with ASD is haloperidol (trade name Haldol), an antipsychotic medication used in the treatment of schizophrenia. Some controlled studies have shown that this drug reduces social withdrawal, stereotyped motor behaviour, and such maladaptive behaviours as self-mutilation and aggression (Anderson et al., 1989; Perry et al., 1989). Many children do not respond positively to the drug, however, and it has not shown any positive effects on other aspects of ASD, such as social functioning and language impairments (Holm & Varley, 1989). Haloperidol also has serious side effects (Posey & McDougle, 2000). In a longitudinal study, over 30 percent of children with ASD developed drug-related dyskinesias, or jerky muscle disturbances, although most such effects went away after the drug was withdrawn (Campbell et al., 1997).

Researchers have also studied naltrexone, an opioid receptor antagonist, and found that this drug reduces hyperactivity in children with ASD and produces a moderate improvement in the initiation of social interactions (Aman & Langworthy, 2000; Willemsen-Swinkels, Buitelaar, Weijnen, & van Engeland, 1995; Williams, Allard, Spears, Dalrymple, & Bloom, 2001). The drug does not appear to affect the core symptoms of ASD, and some evidence suggests that at some doses it may increase self-injurious behaviour (Anderson et al., 1997).

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## SUMMARY

### **10.1 Describe the issues in the diagnosis of psychopathology in children.**

Childhood disorders, like adult disorders, involve a combination of behavioural, cognitive, genetic, neurobiological, and social factors in their aetiology and treatment. The number of children diagnosed with and treated for different psychological disorders has dramatically increased in recent years, but not without controversy. For example, the number of people ever receiving a diagnosis of ADHD (attention-deficit hyperactivity disorder) in Australia and New Zealand is rapidly increasing. Also controversial is the tremendous increase in the number of medication prescriptions given to children. Before making a diagnosis of a particular disorder in children, clinicians must first consider what is typical for a particular age. The children who lie on the floor kicking and screaming when they don't get their way would be assessed differently at age two than at age seven.

The more prevalent childhood disorders are often divided into two broad domains, externalising disorders and internalising disorders. Externalising disorders are characterised by more outward-directed behaviours, such as aggressiveness, non-compliance, overactivity and impulsiveness; the category includes attention-deficit hyperactivity disorder, conduct disorder and oppositional defiant disorder. Internalising disorders are characterised by more inward-focused experiences and behaviours, such as depression, social withdrawal, and anxiety; the category includes childhood anxiety and mood disorders. Children and adolescents may exhibit symptoms from both domains.

### **10.2 Discuss the description, aetiology and treatments for externalising disorders, including ADHD and conduct disorder.**

ADHD and conduct disorder are referred to as externalising disorders. They appear across cultures, although there are also differences in the manifestation of externalising symptoms in different cultures. Both disorders are more common in boys than girls. A number of factors work together to cause ADHD and conduct disorder. Genetic factors play a particularly important role in ADHD but are also implicated in conduct disorder. Neurobiological research has implicated areas of the brain and neurotransmitters such as dopamine in ADHD and amygdala in conduct disorder; neuropsychological deficits are seen in both disorders. Other risk factors for ADHD include low birth weight and maternal smoking. Family and peer variables are also important factors to consider, especially in how they interact with genetic and neurobiological vulnerabilities. The most effective treatment for ADHD is a combination of stimulant medication and behavioural therapy. For conduct disorder, family-based treatments, such as PMT, are effective, as are treatments that include multiple points for intervention, as in MST. Prevention approaches, such as Fast Track, can also be helpful.

### **10.3 Discuss the description, aetiology and treatments for internalising disorders, including depression and anxiety disorders.**

Anxiety disorders and depression in children are referred to as internalising disorders. Depression in childhood and adolescence appears to be similar to depression in adulthood, although there are notable differences. In childhood, depression affects boys and girls equally, but in adolescence girls are affected almost twice as often as boys. Genetics and stressful life events play a role in depression in childhood. Research on cognitive factors in childhood depression supports the notion that attributional style also plays a role; however, this work must consider the developmental stage of the child. A randomised controlled trial found that a combination of medication and CBT was the most effective treatment for depression, but concerns about the effect of medications on suicide risk need to be addressed.

Anxiety and fear are typical in childhood. When fears interfere with functioning, such as keeping a child from school, intervention is warranted. Theories about the causes of anxiety disorders in

children are similar to theories about their causes in adulthood, though less research has been done with children on, for example, cognitive factors. Cognitive-behavioural therapy is an effective intervention for a number of different anxiety disorders in childhood. Other problems, such as PTSD in childhood, require additional study.

**10.4 Understand the learning disorders dyslexia and dyscalculia, and learn the causes and treatments for dyslexia.**

Mental health professionals often refer to specific learning disorders, communication disorders and motor disorders as learning disabilities. Children with dyslexia have significant difficulty with word recognition, reading comprehension and, typically, written spelling as well. Research has uncovered how the brain is involved in dyslexia, particularly areas of the brain that support language, including the temporoparietal and occipitotemporal areas. However, important cultural differences have been noted, suggesting that there may not be one universal mechanism to account for dyslexia. Interventions for dyslexia involve intensive work on reading and language skills.

**10.5 Discuss the description and diagnosis of intellectual disability and the current research on causes and treatments.**

The DSM-5 uses the term *intellectual disability* rather than *mental retardation* and emphasises the importance of assessing intellectual ability and adaptive functioning within a person's cultural group, consistent with the approach of the AAIDD. Categories based on IQ scores will no longer be used. Instead, like the AAIDD, the DSM-5 stresses the importance of identifying an individual's strengths and weaknesses. There are a number of known causes of intellectual disability, including genetic abnormalities, infections and toxins.

**10.6 Describe the symptoms, causes and treatments for autism spectrum disorders.**

Children with autism spectrum disorder (ASD) experience social and emotional disturbances which can make it difficult to maintain and develop relationships. They typically have deficits in nonverbal behaviours in eye-contact, facial expressions and body language. They can display repetitive speech or motor movements, have an excessive adherence to routines and a restricted interest in things that can be viewed as atypical, such as a preoccupation with the moving wheels on a train. They can also have hyper- or hyporeactivity to sensory input or unusual interest in their sensory environment, such as fascination with lights or spinning objects.

Onset is in early childhood. Previously, it was thought that ASD was caused by 'bad parenting'; however, this incorrect belief has been replaced by theories based on evidence that genetic and neurological factors are important in the aetiology.

Psychological treatments currently show the most promise in treating ASD, not medication. Treatments for children with ASD are usually aimed at reducing their unusual behaviour and improving their communication and social skills. In most cases, the earlier the intervention begins, the better the outcome.

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## KEY TERMS

**attention-deficit hyperactivity disorder (ADHD)** a disorder in children marked by difficulties in focusing adaptively on the task at hand, inappropriate fidgeting and antisocial behaviour, and excessive non-goal-directed behaviour

**autism spectrum disorder (ASD)** a diagnostic category in DSM-5 that includes different clinical specifiers relating to severity and extent of language impairment

**childhood onset fluency disorder (stuttering)** communication disorder of childhood marked by frequent and pronounced verbal dysfluencies, such as repetitions of certain sounds

**communication disorders** learning disorders in a child who fails to develop to the degree expected by his or her intellectual level in a specific language skill area; include speech sounds disorder and childhood onset fluency disorder

**conduct disorder** pattern of extreme disobedience in youngsters, including theft, vandalism, lying and early drug use

**developmental psychopathology** the field that studies disorders of childhood within the context of normal life-span development

**Down syndrome (trisomy 21)** a form of intellectual disability caused by a third copy of a particular chromosome; involves an IQ usually less than 50 as well as distinctive physical characteristics

**dyscalculia** learning disorder characterised by difficulty recalling arithmetic facts, counting objects and aligning numbers in columns

**dyslexia** a learning disorder involving significant difficulty with word recognition, reading comprehension and (typically) spelling

**externalising disorders** domain of childhood disorders characterised by outward-directed behaviours, such as aggressiveness, non-compliance, overactivity and impulsiveness; the category includes attention-deficit hyperactivity disorder, conduct disorder and oppositional defiant disorder

**fragile X syndrome** a chromosomal abnormality involving a mutation in the FMR1 gene on the X chromosome

**intellectual disability** a disorder characterised by below-average intellectual functioning associated with impairment in adaptive behaviour and identified at an early age

**internalising disorders** domain of childhood disorders characterised by inward-focused experiences and behaviours, such as depression, social withdrawal and anxiety; the category includes childhood anxiety and mood disorders

**joint attention** interactions between two people require paying attention to each other, whether speaking or communicating emotion nonverbally. This is impaired in children with autism spectrum disorder

**motor disorder** a learning disorder characterised by marked impairment in the development of motor coordination that is not accounted for by a physical disorder such as cerebral palsy

**multisystemic treatment (MST)** treatment for serious juvenile offenders that involves delivering intensive and comprehensive therapy services in the community, targeting the adolescent, the family, the school, and, in some cases, the peer group, in ecologically valid settings and using varied techniques

**parent management training (PMT)** behavioural program in which parents are taught to modify their responses to their children so that prosocial rather than antisocial behaviour is consistently rewarded

**phenylketonuria (PKU)** a genetic deficiency in a liver enzyme, phenylalanine hydroxylase, that causes severe intellectual disability unless phenylalanine can be largely restricted from the diet

**pronoun reversal** a speech problem in which the child refers to himself or herself as 'he,' 'she,' or 'you' and uses 'I' or 'me' in referring to others; often found in the speech of children with autistic disorder

**separation anxiety disorder** a disorder in which the child feels intense fear and distress when away from someone on whom he or she is very dependent

**specific learning disorders** a set of developmental disorders encompassing dyslexia and dyscalculia; characterised by failure to develop in a specific academic area to the degree expected by the child's intellectual level

**speech sounds disorder** communication disorder in childhood in which some words sound like baby talk because the person is not able to make certain speech sounds

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## WEBSITES

1. Department of Health: The Mental Health of Children and Adolescents — an Australian report published in 2015 that is based on a survey of over 6300 families with children aged between the ages of 4 and 17 years. ([www.health.gov.au/internet/main/publishing.nsf/Content/9DA8CA21306FE6EDCA257E2700016945/\\$File/child2.pdf](http://www.health.gov.au/internet/main/publishing.nsf/Content/9DA8CA21306FE6EDCA257E2700016945/$File/child2.pdf))

2. KidsMatter Australia provides information on mental health difficulties for Australian children and families. ([www.kidsmatter.edu.au](http://www.kidsmatter.edu.au))
3. The Conversation: 'Children's mental health needs to be at the heart of school policy' is an article explaining the need for schools to focus more on the mental health of children. (<https://theconversation.com/childrens-mental-health-needs-to-be-at-the-heart-of-school-policy-56091>)
4. The Conversation: 'Poor nutrition can put children at higher risk of mental illness' is an article explaining a possible link between the mental health of children and poor nutrition. (<https://theconversation.com/poor-nutrition-can-put-children-at-higher-risk-of-mental-illness-54836>)

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## REFERENCES

- Achenbach, T. M., Hensley, V. R., Phares, V., & Grayson, D. (1990). Problems and competencies reported by parents of Australian and American children. *Journal of Child Psychology and Psychiatry*, 31, 265–286.
- Adams, K., Halacas, C., Cincotta, M., & Pesich, C. (2014). Mental health and Victorian Aboriginal people: What can data mining tell us? *Australian Journal of Primary Health*, 20(4), 350–355. doi: <http://dx.doi.org/dbgw.lis.curtin.edu.au/10.1071/PY14036>
- Adverse Drug Reactions Advisory Committee. (2014). Use of SSRI antidepressants in children and adolescents, October 2004. Australian Government Department of Health Therapeutic Goods Administration. Retrieved from [www.tga.gov.au/use-ssri-antidepressants-children-and-adolescents-october-2004](http://www.tga.gov.au/use-ssri-antidepressants-children-and-adolescents-october-2004)
- Aman, M. G., & Langworthy, K. (2000). Pharmacotherapy for hyperactivity in children with autism and other pervasive developmental disorders. *Journal of Autism and Developmental Disorders*, 30, 451–459.
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-5)* (5th ed.).
- Anderson, C. A., Hinshaw, S. P., & Simmel, C. (1994). Mother–child interactions in ADHD and comparison boys: Relationships to overt and covert externalizing behavior. *Journal of Abnormal Child Psychology*, 22, 247–265.
- Anderson, L. T., Campbell, M., Adams, P., Small, A. M., Perry, R., & Shell, J. (1989). The effects of haloperidol on discrimination learning and behavioral symptoms in autistic children. *Journal of Autism and Developmental Disorders*, 19, 227–239.
- Anderson, S., Hanson, R., Malecha, M., Oftelie, A., Erickson, C., & Clark, J. M. (1997). The effectiveness of naltrexone in treating task attending, aggression, self-injury, and stereotypic mannerisms of six young males with autism or pervasive developmental disorders. *Journal of Developmental and Physical Disabilities*, 9, 211–221.
- Anthony, J. L., & Lonigan, C. L. (2004). The nature of phonological awareness: Converging evidence from four studies of preschool and early grade school children. *Journal of Educational Psychology*, 96, 43–55.
- Arnold, E. H., O'Leary, S. G., & Edwards, G. H. (1997). Father involvement and self-reported parenting of children with attention deficit hyperactivity disorder. *Journal of Consulting and Clinical Psychology*, 65, 337–342.
- Arnold, L. E., Elliott, M., Sachs, L., et al. (2003). Effects of ethnicity on treatment attendance, stimulant response/dose, and 14-month outcome in ADHD. *Journal of Consulting and Clinical Psychology*, 71, 713–727.
- Australian Bureau of Statistics (2015). *National health survey: first results, 2014–15*, cat. no. 4364.0.55.001. Canberra, ACT.
- Australian Bureau of Statistics. (2012). *Autism in Australia*, cat. no. 4428.0. Retrieved from [www.abs.gov.au/AUSSTATS/abs@.nsf/Latestproducts/4428.0Main%20Features32012](http://www.abs.gov.au/AUSSTATS/abs@.nsf/Latestproducts/4428.0Main%20Features32012)
- Autism NZ. (n.d.). *About autism*. Retrieved from [www.autismnz.org.nz/about\\_autism](http://www.autismnz.org.nz/about_autism)
- Axelson, D. A., Birmaher, B., Findling, R. L., Fristad, M. A., Kowatch, R. A., Youngstrom, E. A., et al. (2011). Concerns regarding the inclusion of temper dysregulation disorder with dysphoria in the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition. *Journal of Clinical Psychiatry*, 72(9), 1257–1262.
- Bailey, A., LeCouteur, A., Gottesman, I., Bolton, P., Simonoff, E., Yuzda, E., & Rutter, M. (1995). Autism as a strongly genetic disorder: Evidence from a British twin study. *Psychological Medicine*, 25, 63–77.
- Banich, M. T., Passarotti, A. M., White, D. A., Nortz, M. J., & Steiner, R. D. (2000). Interhemispheric interaction during childhood: II. Children with early-treated phenylketonuria. *Developmental Neuropsychology*, 18, 53–71.
- Barber, C. (2008). *Comfortably numb*. New York: Pantheon Books.
- Barkley, R. A. (1990). *Attention-deficit hyperactivity disorder: A handbook for diagnosis and treatment*. New York: Guilford Press.
- Barkley, R. A., DuPaul, G. J., & McMurray, M. B. (1990). A comprehensive evaluation of attention deficit disorder with and without hyperactivity defined by research criteria. *Journal of Consulting and Clinical Psychology*, 58, 775–789.
- Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2002). The persistence of attention-deficit hyperactivity disorder into young adulthood as a function of reporting source and definition of disorder. *Journal of Abnormal Psychology*, 111, 279–289.
- Barkley, R. A., Fischer, M., Smallish, L., & Fletcher, K. (2003). Does the treatment of attention-deficit/hyperactivity disorder with stimulants contribute to drug use/abuse? A 13 year prospective study. *Pediatrics*, 111, 97–109.
- Barkley, R. A., Grodzinsky, G., & DuPaul, G. J. (1992). Frontal lobe functions in attention deficit disorder with and without hyperactivity: A review and research report. *Journal of Abnormal Child Psychology*, 20, 163–188.

- Barkley, R. A., Karlsson, J., & Pollard, S. (1985). Effects of age on the mother-child interactions of hyperactive children. *Journal of Abnormal Child Psychology*, 13, 631–638.
- Beauchaine, T. P., Hinshaw, S. P., & Pang, K. L. (2010). Comorbidity of attention-deficit/hyperactivity disorder and early-onset conduct disorder: Biological, environmental, and developmental mechanisms. *Clinical Psychology: Science and Practice*, 17, 327–336.
- Benjamin, C. L., Harrison, J. P., Settiani, C. A., Brodman, D. M., & Kendall, P. C. (2013). Anxiety and related outcomes in young adults 7 to 19 years after receiving treatment for child anxiety. *Journal of Consulting and Clinical Psychology*, 81(5), 865–876.
- Bhutta, A. T., Cleves, M. A., Casey, P. H., Cradock, M. M., & Anand, K. J. (2002). Cognitive and behavioral outcomes of school-aged children who were born preterm: A meta-analysis. *Journal of the American Medical Association*, 288, 728–737.
- Biederman, J., & Faraone, S. (2004). The Massachusetts General Hospital studies of gender influences on attention-deficit/hyperactivity disorder in youth and relatives. *Psychiatric Clinics of North America*, 27, 215–224.
- Biederman, J., Mick, E., Faraone, S. V., Spencer, T., Wilens, T. E., & Wozniak, J. (2000). Pediatric mania: A developmental subtype of bipolar disorder? *Biological Psychiatry*, 48, 458–466.
- Biederman, J., Monuteau, M. C., Mick, E., Spencer, T., Wilens, T. E., Silva, J. M., et al. (2006). Young adult outcome of attention deficit hyperactivity disorder: a controlled 10-year follow-up study. *Psychological Medicine*, 36, 167–179.
- Biederman, J., Petty, C. R., Monuteaux, M. C., Fried, R., Byrne, D., Mirto, T., et al. (2010). Adult psychiatric outcomes of girls with attention deficit hyperactivity disorder: 11-year follow-up in a longitudinal case-control study. *American Journal of Psychiatry*, 167, 409–417.
- Blachman, D. R., & Hinshaw, S. P. (2002). Patterns of friendship among girls with and without attention-deficit/hyperactivity disorder. *Journal of Abnormal Child Psychology*, 30, 625–640.
- Blair, R. J. (2013). The neurobiology of psychopathic traits in youths. *Nature Reviews Neuroscience*, 14(11), 786–799.
- Boegels, S. M., & Zigterman, D. (2000). Dysfunctional cognitions in children with social phobia, separation anxiety disorder, and generalized anxiety disorder. *Journal of Abnormal Child Psychology*, 28, 205–211.
- Boets, B., Op de Beeck, H. P., Vandermosten, M., Scott, S. K., Gillebert, C. R., Mantini, D., et al. (2013). Intact but less accessible phonetic representations in adults with dyslexia. *Science*, 342(6163), 1251–1254.
- Borduin, C. M., Mann, B. J., Cone, L. T., Henggeler, S. W., Fucci, B. R., Blaske, D. M., & Williams, R. A. (1995). Multisystemic treatment of serious juvenile offenders: Long-term prevention of criminality and violence. *Journal of Consulting and Clinical Psychology*, 63, 569–578.
- Bosquet, M., & Egeland, B. (2006). The development and maintenance of anxiety symptoms from infancy through adolescence in a longitudinal sample. *Development and Psychopathology*, 18, 517–550.
- Braswell, L., & Kendall, P. C. (1988). Cognitive-behavioral methods with children. In K. S. Dobson (Ed.), *Handbook of Cognitive-Behavioral Therapies*. New York: Guilford.
- Braun, J. M., Kahn, R. S., Froehlich, T., Auinger, P., & Lanphear, B. (2006). Exposures to environmental toxicants and attention deficit hyperactivity disorder in U.S. children. *Environmental Health Perspectives*, 114, 1904–1909.
- Breslau, N., Brown, G. G., Del Dotto, J. E., Kumar, S., Ezhuthachan, S., Andreski, P., & Hufnagle, K. G. (1996). Psychiatric sequelae of low birth weight at 6 years of age. *Journal of Abnormal Child Psychology*, 24, 385–400.
- Bridge, J. A., Iyengar, S., Salary, C. B., et al. (2007). Clinical response and risk for reported suicidal ideation and suicide attempts in pediatric antidepressant treatment: A meta-analysis of randomized controlled trials. *Journal of the American Medical Association*, 297, 1683–1696.
- Brookes, K., Mill, J., Guindalini, C., Curran, S., Xu, X., Knight, J., Chen, C. K., Huang, Y. S., Senth, V., Taylor, E., Chen, W., Breen, G., & Asherson, P. (2006). A common haplotype of the dopamine transporter gene associated with attention-deficit/hyperactivity disorder and interacting with maternal use of alcohol during pregnancy. *Archives of General Psychiatry*, 63, 74–81.
- Burt, S. A. (2009). Are there meaningful etiological differences within antisocial behavior? Results of a meta-analysis. *Clinical Psychology Review*, 29(2), 163–178.
- Campbell, M., Armenteros, J. L., Malone, R. P., Adams, P. B., Eisenberg, Z. W., & Overall, J. E. (1997). Neuroleptic-related dyskinesias in autistic children: A prospective, longitudinal study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 835–843.
- Capaldi, D. M., & Patterson, G. R. (1994). Interrelated influences of contextual factors on antisocial behavior in childhood and adolescence for males. In D. C. Fowles, P. Sutker & S. H. Goodman (Eds.), *Progress in experimental personality and psychopathology research* (pp. 165–198). New York: Springer-Verlag.
- Capps, L., Losh, M., & Thurber, C. (2000). “The frog ate the bug and made his mouth sad”: Narrative competence in children with autism. *Journal of Abnormal Child Psychology*, 28, 193–204.
- Capps, L., Rasco, L., Losh, M., & Heerey, E. (1999). *Understanding of self-conscious emotions in high-functioning children with autism*. Paper presented at the Biennial Meeting of the Society for Research In Child Development, Albuquerque, NM.
- Capps, L., Yirmiya, N., & Sigman, M. (1992). Understanding of simple and complex emotion in high-functioning children with autism. *Journal of Child Psychology and Psychiatry*, 33, 1169–1182.
- Carlson, G. A., & Meyer, S. E. (2006). Phenomenology and diagnosis of bipolar disorder in children, adolescents, and adults: Complexities and developmental issues. *Development and Psychopathology* 18, 939–969.

- Casey, J. E., Rourke, B. P., & Del Dotto, J. E. (1996). Learning disabilities in children with attention deficit disorder with and without hyperactivity. *Child Neuropsychology*, 2, 83–98.
- Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 297, 851–854.
- Castellanos, F. X., Lee, P. P., Sharp, W., Jeffries, N. O., Greenstein, D. K., et al. (2002). Developmental trajectories of brain volume abnormalities in children and adolescents with attention-deficit/hyperactivity disorder. *Journal of the American Medical Association*, 288, 1740–1748.
- Centers for Disease Control and Prevention (CDC). (2014). *HIV Surveillance Report: Diagnoses of HIV infection and AIDS in the United States and dependent areas, 2011*. Atlanta: CDC Retrieved from [www.cdc.gov/hiv/surveillance/resources/reports](http://www.cdc.gov/hiv/surveillance/resources/reports)
- Chronis, A. M., Jones, H. A., & Raggi, V. L. (2006). Evidence-based psychosocial treatments for children and adolescents with attention-deficit/hyperactivity disorder. *Clinical Psychology Review*, 26, 486–502.
- Cimbor, D. M., & McIntosh, D. N. (2003). Emotional responses to antisocial acts in adolescent males with conduct disorder: A link to affective morality. *Journal of Clinical Child and Adolescent Psychology*, 32, 296–301.
- Coie, J. D., & Dodge, K. A. (1998). Aggression and antisocial behavior. In W. Damon & N. Eisenberg (Eds.), *Handbook of child psychology: Volume 3: Social, emotional and personality development* (pp. 779–862). New York: John Wiley & Sons.
- Cole, D. A., Ciesla, J. A., Dallaire, D. H. et al. (2008). Emergence of attributional style and its relations to depressive symptoms. *Journal of Abnormal Psychology*, 117, 16–31.
- Cole, D. A., Martin, J. M., Peeke, L. G., Seroczynski, A. D., & Hoffman, K. (1998). Are cognitive errors of underestimation predictive or reflective of depressive symptoms in children? A longitudinal study. *Journal of Abnormal Psychology*, 107, 481–496.
- Commonwealth of Australia (2013). *National Aboriginal and Torres Strait Islander health plan 2013–2023* (Publication approval number 10290). Canberra, ACT.
- Conduct Problems Prevention Research Group. (2010a). The difficulty of maintaining positive intervention effects: A look at disruptive behavior, deviant peer relations, and social skills during the middle school years. *Journal of Early Adolescence*, 30(4).
- Conduct Problems Prevention Research Group. (2010b). Fast track intervention effects on youth arrests and delinquency. *Journal of Experimental Criminology*, 6(2), 131–157.
- Conduct Problems Prevention Research Group. (2011). The effects of the fast track preventive intervention on the development of conduct disorder across childhood. *Child Development*, 82(1), 331–345.
- Constantino, J. N., Zhang, Z., Frazier, T., Abbachi, A. M., & Law, P. (2010). Sibling recurrence and the genetic epidemiology of autism. *American Journal of Psychiatry*, 167, 1349–1356.
- Copeland, W. E., Angold, A., Costello, E. J., & Egger, H. (2013). Prevalence, comorbidity, and correlates of DSM-5 proposed disruptive mood dysregulation disorder. *American Journal of Psychiatry*, 170(2), 173–179.
- Cortese, S., Kelly, C., Chabernaud, C., Proal, E., Di Martino, A., Milham, M. P., & Castellanos, F. X. (2012). Toward systems neuroscience of ADHD: A meta-analysis of 55 fMRI studies. *American Journal of Psychiatry*, 169(10), 1038–1055.
- Costello, J. E., Erkanli, A., & Angold, A. (2006). Is there an epidemic of child or adolescent depression? *Journal of Child Psychology and Psychiatry*, 47(12), 1263–1271.
- Courchesne, E. (2004). Brain development in autism: Early overgrowth followed by premature arrests of growth. *Mental Retardation and Developmental Disabilities Research Reviews*, 10, 106–111.
- Courchesne, E., Carnes, B. S., & Davis, H. R. (2001). Unusual brain growth patterns in early life in patients with autistic disorder: An MRI study. *Neurology*, 57, 245–254.
- Courchesne, E., Carper, R., & Akshoomoff, N. (2003). Evidence of brain overgrowth in the first year of life in autism. *Journal of the American Medical Association*, 290, 337–344.
- Critchley, H. D., Daly, E. M., Bullmore, E. T., Williams, S. C. R., Van Amelsvoort, T., Robertson, D. M., Rowe, A., Phillips, M., McAlonan, G., Howlin, P., & Murphy, D. G. M. (2001). The functional neuroanatomy of social behaviour: Changes in cerebral blood flow when people with autistic disorder process facial expressions. *Brain*, 123, 2203–2212.
- Crozier, J. C., Dodge, K. A., Griffith, R. et al. (2008). Social information processing and cardiac predictors of adolescent antisocial behavior. *Journal of Abnormal Psychology*, 117, 253–267.
- Cummings, C. M., Caporino, N. E., & Kendall, P. C. (2014). Comorbidity of anxiety and depression in children and adolescents: 20 years after. *Psychological Bulletin*, 140(3), 816–845.
- Curry, J. F. (2001). Specific psychotherapies for childhood and adolescent depression. *Biological Psychiatry*, 49, 1091–1100.
- Curry, J., Silva, S., Rohde, P., Ginsburg, G., Kratochvil, C., Simons, A., Kirchner, J., May, D., Kennard, B., et al. (2011). Recovery and recurrence following treatment for adolescent major depression. *Archives of General Psychiatry*, 68, 263–270.
- Davies, D. K., Stock, S. E., & Wehmeyer, M. (2003). Application of computer simulation to teach ATM access to individuals with intellectual disabilities. *Education and Training in Developmental Disabilities*, 38, 451–456.
- Davis, L., & Siegel, L. J. (2000). Posttraumatic stress disorder in children and adolescents: A review and analysis. *Clinical Child and Family Psychology Review*, 3, 135–153.
- Davis, T. E., May, A., & Whiting, S. E. (2011). Evidence-based treatment of anxiety and phobia in children and adolescents: Current status and effects on the emotional response. *Clinical Psychology Review*, 31, 592–602.
- Dawson, G., Toth, K., Abbott, R., Osterling, J., Munson, J., Estes, A., & Liaw, J. (2004). Early social attention impairments in autism: Social orienting, joint attention, and attention to distress. *Developmental Psychology*, 40, 271–283.

- Dickstein, D. P., & Leibenluft, E. (2006). Emotion regulation in children and adolescents: Boundaries between normalcy and bipolar disorder. *Development and Psychopathology*, 18, 1105–1131.
- Dishion, T. J., & Andrews, D. W. (1995). Preventing escalation in problem behaviors with high-risk young adolescents: Immediate and 1-year outcomes. *Journal of Consulting and Clinical Psychology*, 63, 538–548.
- Dishion, T. J., Patterson, G. R., & Kavanagh, K. A. (1992). An experimental test of the coercion model: Linking theory, measurement, and intervention. In J. McCord & R. E. Tremblay (Eds.), *Preventing Antisocial Behavior* (pp. 253–282). New York: Guilford Press.
- Dodge, K. A., & Frame, C. L. (1982). Social cognitive biases and deficits in aggressive boys. *Child Development*, 53, 620–635.
- Dodge, K. A., & Godwin, J. (2013). Social-information-processing patterns mediate the impact of preventive intervention on adolescent antisocial behavior. *Psychological Science*, 24(4), 456–465.
- Duff, J. (n.d.) Why the increase in Autism (ASD), ADHD and neurodevelopmental disorders? *Australian Autism ADHD Foundation*. Retrieved from [www.autism-adhd.org.au/why-is-adhd-increasing](http://www.autism-adhd.org.au/why-is-adhd-increasing)
- Egger, H. L., & Angold, A. (2006). Common emotional and behavioral disorders in preschool children: presentation, nosology, and epidemiology. *Journal of Child Psychology and Psychiatry*, 47(3–4), 313–337.
- Elkins, I., McGue, M., & Iacono, W. (2007). Prospective effects of attention-deficit/hyperactivity disorder, conduct disorder, and sex on adolescent substance use and abuse. *Archives of General Psychiatry*, 64, 1145–1152.
- Erhardt, D., & Hinshaw, S. P. (1994). Initial sociometric impressions of attention-deficit hyperactivity disorder and comparison boys: Predictions from social behaviors and nonverbal behaviors. *Journal of Consulting and Clinical Psychology*, 62, 833–842.
- Essex, M. J., Klein, M. H., Slattey, M. J., Goldsmith, H. H., & Kalin, N. H. (2010). Early risk factors and developmental pathways to chronic high inhibition and social anxiety disorder in adolescence. *American Journal of Psychiatry*, 167, 40–46.
- Express Scripts Lab. (2014). Turning attention to ADHD report: An express scripts report on U.S. medication trends for attention deficit hyperactivity disorder.
- Faraone, S. V., Biederman, J., & Mick, E. (2005). The age-dependent decline of attention deficit hyperactivity disorder: A meta-analysis of follow-up studies. *Psychological Medicine*, 36, 159–165.
- Faraone, S. V., Biederman, J., Weber, W., & Russell, R. L. (1998). Psychiatric, neuropsychological, and psychosocial features of DSM-IV subtypes of attention-deficit/hyperactivity disorder: Results from a clinically referred sample. *Journal of the American Academy of Child and Adolescent Psychiatry*, 37, 185–193.
- Faraone, S. V., Sergeant, J., Gillberg, C., & Biederman, J. (2003). The worldwide prevalence of ADHD: is it an American condition? *World Psychiatry*, 2(2), 104–113. Retrieved from [www.ncbi.nlm.nih.gov/pmc/articles/PMC1525089/table/T6/](http://www.ncbi.nlm.nih.gov/pmc/articles/PMC1525089/table/T6/)
- Feingold, B. F. (1973). *Introduction to clinical allergy*. Springfield, IL: Charles C. Thomas.
- Fishler, K., Azen, C. G., Henderson, R., Friedman, E. G., & Koch, R. (1987). Psychoeducational findings among children treated for phenylketonuria. *American Journal of Mental Deficiency*, 92, 65–73.
- Fontaine, N. M. G., McCrory, E. J. P., Boivin, M., Moffitt, T. E., & Viding, E. (2011). Predictors and outcomes of joint trajectories of callous-unemotional traits and conduct problems in childhood. *Journal of Abnormal Psychology*, 120, 730–742.
- Forero, D. A., Arboleda, G. H., Vasquez, R., & Arboleda, H. (2009). Candidate genes involved in neural plasticity and the risk for attention-deficit hyperactivity disorder: A meta-analysis of 8 common variants. *Journal of Psychiatry and Neuroscience*, 34(5), 361–366.
- Franklin, M. E., Sapyta, J., Freeman, J. B., Khanna, M., Compton, S., Almirall, D., et al. (2011). Cognitive behavior therapy augmentation of pharmacotherapy in pediatric obsessive-compulsive disorder: The Pediatric OCD Treatment Study II (POTS II) randomized controlled trial. *Journal of the American Medical Association*, 306(11), 1224–1232.
- Freeman, J., Sapyta, J., Garcia, A., & et al. (2014). Family-based treatment of early childhood obsessive-compulsive disorder: The pediatric obsessive-compulsive disorder treatment study for young children (POTS jr)—a randomized clinical trial. *Journal of the American Medical Association*, 306(11), 1224–1232.
- Frick, P. J., Ray, J. V., Thornton, L. C., & Kahn, R. E. (2014). Can callous-unemotional traits enhance the understanding, diagnosis, and treatment of serious conduct problems in children and adolescents? A comprehensive review. *Psychological Bulletin*, 140(1), 1–57.
- Friend, A., DeFries, J. C., Olson, R. K., & Pennington, B. F. (2009). Heritability of high reading ability and its interaction with parental education. *Behavior Genetics*, 39, 427–436.
- Garber, J. (2006). Depression in children and adolescents: Linking risk research and prevention. *American Journal of Preventative Medicine*, 31, 5104–5125.
- Garber, J., Clarke, G. N., Weersing, V. R., Beardslee, W. R., Brent, D. A., Gladstone, T. R., et al. (2009). Prevention of depression in at-risk adolescents: A randomized controlled trial. *Journal of the American Medical Association*, 301, 2215–2224.
- Garber, J., & Flynn, C. (2001). Vulnerability to depression in childhood and adolescence. In R. M. Ingram & J. M. Price (Eds.), *Vulnerability to psychopathology: Risk across the lifespan* (pp. 175–225). New York: Guilford Press.
- Garber, J., Kelly, M. K., & Martin, N. C. (2002). Developmental trajectories of adolescents' depressive symptoms: Predictors of change. *Journal of Consulting and Clinical Psychology*, 70, 79–95.
- Gaus, V. L. (2007). *Cognitive behavior therapy for adults with Asperger's syndrome*. New York: Guilford Press.
- Gernsbacher, M. A., Dawson, M., & Goldsmith, H. H. (2005). Three reasons not to believe in an autism epidemic. *Current Directions in Psychological Science*, 14, 55–58.
- Gizer, I. R., Ficks, C., & Waldman, I. D. (2009). Candidate gene studies of ADHD: A meta-analytic review. *Human Genetics*, 126(1), 51–90.

- Gopnik, A., Capps, L., & Meltzoff, A. N. (2000). Early theories of mind: What the theory theory can tell us about autism. In S. Baron-Cohen, H. Tager-Flusberg & D. Cohen (Eds.), *Understanding Other Minds* (2nd ed., pp. 50–72). Oxford, UK: Oxford University Press.
- Grandin, T. (1986). *Emergence: Labeled autistic*. Novato, CA: Arena Press.
- Grandin, T. (1995). *Thinking in pictures*. New York: Doubleday.
- Grandin, T. (2008). *The way I see it: A personal look at autism and Asperger's*. Arlington, TX: Future Horizons.
- Grandin, T. (2013). *The autistic brain*. New York: Houghton Mifflin Harcourt.
- Hagerman, R. (2006). Lessons from fragile x regarding neurobiology, autism, and neurodegeneration. *Developmental and Behavioral Pediatrics*, 27, 63–74.
- Hallmayer, J., Cleveland, S., Torres, A., Phillips, J., Cohen, B., Torigoe, T., Miller, J., Fedele, A., et al. (2011). Genetic heritability and shared environmental factors among twin pairs with autism. *Archives of General Psychiatry*, 68, 1095–1102.
- Hammen, C. (2009). Adolescent depression: Stressful interpersonal contexts and risk for recurrence. *Current Directions in Psychological Science*, 18, 200–204.
- Hammen, C., & Brennan, P. (2001). Depressed adolescents of depressed and nondepressed mothers: Tests of an interpersonal impairment hypothesis. *Journal of Consulting and Clinical Psychology*, 69, 284–294.
- Hamsheer, M. L., Langley, K., Martin, J., Agha, S. S., Stergiakouli, E., Anney, R. J., et al. (2013). High loading of polygenic risk for ADHD in children with comorbid aggression. *American Journal of Psychiatry*, 170(8), 909–916.
- Hankin, B. J., Abramson, L. Y., Moffitt, T. E., Silva, P. A., McGee, R., et al. (1998). Development of depression from preadolescence to young adulthood: Emerging gender differences in a 10-year longitudinal study. *Journal of Abnormal Psychology*, 107, 128–140.
- Hart, E. L., Lahey, B. B., Loeber, R., Applegate, B., & Frick, P. J. (1995). Developmental changes in attention-deficit hyperactivity disorder in boys: A four-year longitudinal study. *Journal of Abnormal Child Psychology*, 23, 729–750.
- Hazel, N. A., Hamman, C., Brennan, P. A., Najman, J. (2008). Early childhood adversity and adolescent depression: The mediating role of continued stress. *Psychological Medicine*, 38, 581–589.
- Hazlett, H. C., Poe, M. D., Gerig, G., Styner, M., Chappell, C., Smith, R. G., Vachet, C., & Priven, J. (2011). Early brain overgrowth in autism associated with an increase in cortical surface area before age 2 years. *Archives of General Psychiatry*, 68, 467–476.
- Heller, T. L., Baker, B. L., Henker, B., & Hinshaw, S. P. (1996). Externalizing behavior and cognitive functioning from preschool to first grade: Stability and predictors. *Journal of Clinical Child Psychology*, 25, 376–387.
- Henggeler, S. W., Schoenwald, S. D., Borduin, C. M., Rowland, M. D., & Cunningham, P. B. (1998). *Multisystemic treatment of antisocial behavior in children and adolescents*. New York: Guilford Press.
- Hinshaw, S. P. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: I. Background characteristics, comorbidity, cognitive and social functioning, and parenting practices. *Journal of Consulting and Clinical Psychology*, 70, 1086–1098.
- Hinshaw, S. P., Carte, E. T., Sami, N., Treuting, J. J., & Zupan, B. A. (2002). Preadolescent girls with attention-deficit/hyperactivity disorder: II. Neuropsychological performance in relation to subtypes and individual classification. *Journal of Consulting and Clinical Psychology*, 70, 1099–1111.
- Hinshaw, S. P., & Lee, S. S. (2003). Oppositional defiant and conduct disorders. In E. J. Mash & R. A. Barkley (Eds.), *Child Psychopathology* (2nd ed., pp. 144–198). New York: Guilford Press.
- Hinshaw, S. P., & Melnick, S. M. (1995). Peer relationships in boys with attention-deficit hyperactivity disorder with and without comorbid aggression. *Development and Psychopathology*, 7, 627–647.
- Hinshaw, S. P., Owens, E. B., Sami, N., & Fargeon, S. (2006). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into adolescence: Evidence for continuing cross-domain impairment. *Journal of Consulting and Clinical Psychology*, 74, 489–499.
- Hinshaw, S. P., Owens, E. B., Zalecki, C., Huggins, S. P., Montenegro-Nevado, A. J., Schrodek, E., & Swanson, E. N. (2012). Prospective follow-up of girls with attention-deficit/hyperactivity disorder into early adulthood: Continuing impairment includes elevated risk for suicide attempts and self-injury. *Journal of Consulting and Clinical Psychology*, 80(6), 1041–1051.
- Hinshaw, S. P., & Scheffler, R. M. (2014). *The ADHD explosion: Myths, medication, money, and today's push for performance*. New York: Oxford University Press.
- Hinshaw, S. P., Zupan, B. A., Simmel, C., Nigg, J. T., & Melnick, S. (1997). Peer status in boys with and without attention-deficit hyperactivity disorder: Predictions from overt and covert antisocial behavior, social isolation, and authoritative parenting beliefs. *Child Development*, 68, 880–896.
- Hobson, R. P., & Lee, A. (1998). Hello and goodbye: A study of social engagement in autism. *Journal of Autism and Developmental Disorders*, 28, 117–127.
- Holm, V. A., & Varley, C. K. (1989). Pharmacological treatment of autistic children. In G. Dawson (Ed.), *Autism: Nature, diagnosis, and treatment* (pp. 386–404). New York: Guilford Press.
- Horowitz, J. L., & Garber, J. (2006). The prevention of depressive symptoms in children and adolescents: A meta-analytic review. *Journal of Consulting and Clinical Psychology*, 74, 401–415.
- Howlin, P., Goode, S., Hutton, J., & Rutter, M. (2004). Adult outcome of children with autism. *Journal of Child Psychology and Psychiatry*, 45, 212–229.
- Howlin, P., Mawhood, L., & Rutter, M. (2000). Autism and developmental receptive language disorder—A follow-up comparison in early adult life. II. Social, behavioral, and psychiatric outcomes. *Journal of Child Psychiatry and Psychology*, 41, 561–578.

- Hoza, B., Murray-Close, D., Arnold, L. E., Hinshaw, S. P., Hechtmen, L., & The MTA Cooperative Group. (2010). Time-dependent changes in positive illusory self-perceptions of children with attention-deficit/hyperactivity disorder: A developmental psychopathology perspective. *Developmental and Psychopathology*, 22, 375–390.
- Hughes, C., & Agran, M. (1993). Teaching persons with severe disabilities to use self-instruction in community settings: An analysis of applications. *Journal of the Association for Persons with Severe Handicaps*, 18, 261–274.
- Hughes, C., Hugo, K., & Blatt, J. (1996). Self-instructional intervention for teaching generalized problem-solving within a functional task sequence. *American Journal on Mental Retardation*, 100, 565–579.
- Huijbregts, S. C. J., de Sonnevile, L. M. J., Licht, R., van Spronsen, F. J., Verkerk, P. H., & Sergeant, J. A. (2002). Sustained attention and inhibition of cognitive interference in treated phenylketonuria: Associations with concurrent and lifetime phenylalanine concentrations. *Neuropsychologia*, 40, 7–15.
- Institute of Medicine. (2004). *Immunization safety review: Vaccines and autism*. Immunization Safety Review Board on Health Promotion and Disease Prevention. Washington, DC: National Academies Press.
- James, A., Hoang, U., Seagroatt, V., Clacey, J., Goldacre, M., & Leibenluft, E. (2014). A comparison of American and English hospital discharge rates for pediatric bipolar disorder, 2000 to 2010. *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(6), 614–624.
- Jensen, P. S., Arnold, L. E., Swanson, J. M. et al. (2007). 3-year follow-up of the NIMH MTA study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 989–1002.
- Jensen, P. S., Martin, D., & Cantwell, D. P. (1997). Comorbidity in ADHD: Implications for research, practice, and DSM-V. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 1065–1079.
- Johnston, C., & Marsh, E. J. (2001). Families of children with attention-deficit/hyperactivity disorder: Review and recommendations for future research. *Clinical Child and Family Psychology Review*, 4, 183–207.
- Jones, W., & Klin, A. (2013). Attention to eyes is present but in decline in 2-6-month-old infants later diagnosed with autism. *Nature*, 504(7480), 427–431.
- Jordan, N. C. (2007). Do words count? Connections between mathematics and reading difficulties. In D. B. Berch & M. M. M. Mazzocco (Eds.), *Why is math so hard for some children?* (pp. 107–120). Baltimore: Brooks.
- Kanner, L. (1943). Autistic disturbances of affective contact. *Nervous Child*, 2, 217–250.
- Kasari, C., Freeman, S., & Paparella, T. (2006). Joint attention and symbolic play in young children with autism: A randomized controlled intervention study. *Journal of Child Psychology and Psychiatry*, 47, 611–620.
- Kasari, C., Paparella, T., Freeman, S., & Jahromi, L. B. (2008). Language outcome in autism: Randomized comparison of joint attention and play interventions. *Journal of Consulting and Clinical Psychology*, 76, 125–137.
- Kazdin, A. E. (2005). *Parent management training: Treatment for oppositional, aggressive, and antisocial behavior in children and adolescents*. New York: Oxford University Press.
- Kendall, P. C., Aschenbrand, S. G., & Hudson, J. L. (2003). Child-focused treatment of anxiety. In A. E. Kazdin & J. R. Weisz (Eds.), *Evidence-based psychotherapies for children and adolescents* (pp. 81–100). New York: Guilford Press.
- Kendall, P. C., Flannery-Schroeder, E. C., Panichelli-Mindel, S., Southam-Gerow, M., Henin, A., & Warman, M. (1997). Therapy for youths with anxiety disorders: A second randomized clinical trial. *Journal of Consulting and Clinical Psychology*, 65, 366–380.
- Kendall, P. C., Hudson, J. L., Gosch, E., Flannery-Schroeder, E., & Suveg, C. (2008). Cognitive-behavioral therapy for anxiety disordered youth: A randomized clinical trial evaluating child and family modalities. *Journal of Consulting and Clinical Psychology*, 76, 282–297.
- Kendall, P. C., Safford, S., Flannery-Schroeder, E., & Webb, A. (2004). Child anxiety treatment: Outcomes in adolescence and impact on substance use and depression at 7.4-year follow-up. *Journal of Consulting and Clinical Psychology*, 72, 276–287.
- Kendler, K. S., Jacobson, K., Myers, J. M., & Eaves, L. J. (2008). A genetically informative developmental study of the relationship between conduct disorder and peer deviance in males. *Psychological Medicine*, 38, 1001–1011.
- Klein, D. N., Lewinsohn, P. M., Seeley, J. R., & Rohde, P. A. (2001). A family study of major depressive disorder in a community sample of adolescents. *Archives of General Psychiatry*, 58, 13–20.
- Kremen, W. S., Jacobson, K. C., Xian, H., Eisen, S. A., Waterman, B., Toomey, R., et al. (2005). Heritability of word recognition in middle-aged men varies as a function of parental education. *Behavior Genetics*, 35, 417–433.
- Kurian, B. T., Ray, W. A., Arbogast, P. G., Fuchs, D. C., Dudley, J. A., & Cooper, W. O. (2007). Effect of regulatory warnings on antidepressant prescribing for children and adolescents. *Archives of General Psychiatry*, 161, 690–696.
- Lahey, B. B., Loeber, R., Hart, E. L., Frick, P. J., Applegate, B., Zhang, Q., et al. (1995). Four year longitudinal study of conduct disorder in boys: Patterns and predictors of persistence. *Journal of Abnormal Psychology*, 104, 83–93.
- Lahey, B. B., McBurnett, K., & Loeber, R. (2000). Are attention-deficit/hyperactivity disorder and oppositional defiant disorder developmental precursors to conduct disorder? In A. J. Sameroff & M. Lewis, et al. (Eds.), *Handbook of developmental psychology* (2nd ed., pp. 431–446). New York: Kluwer Academic/Plenum.
- Lahey, B. B., Van Hulle, C. A., Singh, A. L., Waldman, I. D., & Rathouz, P. J. (2011). Higher-order genetic and environmental structure of prevalent forms of child and adolescent psychopathology. *Archives of General Psychiatry*, 68(2), 181–189.
- Lahey, B. B., & Waldman, I. D. (2012). Phenotypic and causal structure of conduct disorder in the broader context of prevalent forms of psychopathology. *Journal of Child Psychology and Psychiatry*, 53, 536–557.
- Landa, R. J., Holman, K. C., & Garrett-Mayer, E. (2007). Social and communication development in toddlers with early and later diagnosis of autism spectrum disorders. *Archives of General Psychiatry*, 64, 853–864.

- Landerl, K., Fussenegger, B., Moll, K., & Willburger, E. (2009). Dyslexia and dyscalculia: Two learning disorders with different cognitive profiles. *Journal of Experimental Child Psychology*, 103, 309–324.
- Lau, J. Y. F., Gregory, A. M., Goldwin, M. A., Pine, D. S., & Eley, T. C. (2007). Assessing gene–environment interactions on anxiety symptom subtypes across childhood and adolescence. *Development and Psychopathology*, 19, 1129–1146.
- Lawrence, D., Johnson, S., Hafekost, J., Boterhoven de Haan, K., Sawyer, M., Ainley, J., & Zubrick, S. R. (2015). *The mental health of children and adolescents (pp. 23–64). Report on the second Australian child and adolescent survey of mental health and wellbeing*. Department of Health, Canberra. Retrieved from [www.health.gov.au/internet/main/publishing.nsf/Content/9DA8CA21306FE6EDCA257E2700016945/\\$File/pt2.pdf](http://www.health.gov.au/internet/main/publishing.nsf/Content/9DA8CA21306FE6EDCA257E2700016945/$File/pt2.pdf)
- Le Couteur, A., Bailey, A., Goode, S., Pickles, A., Robertson, S., Gottesman, I., & Rutter, M. (1996). A broader phenotype of autism: The clinical spectrum in twins. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 37, 785–801.
- Lee, S. S., Lahey, B., Owens, E. B., & Hinshaw, S. P. (2008). Few preschool boys and girls with ADHD are well-adjusted during adolescence. *Journal of Abnormal Child Psychology*, 36, 373–383.
- Leibenluft, E. (2011). Severe mood dysregulation, irritability, and the diagnostic boundaries of bipolar disorder in youths. *American Journal of Psychiatry*, 168(2), 129–142.
- Levy, F., Hay, D. A., McStephen, M., Wood, C., & Waldman, I. (1997). Attention-deficit hyperactivity disorder: A category or a continuum? Genetic analysis of a large-scale twin study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 737–744.
- Lewinsohn, P. M., & Clarke, G. N. (1999). Psychosocial treatments for adolescent depression. *Clinical Psychology Review*, 19, 329–342.
- Lewinsohn, P. M., Petit, J. W., Joiner, T. E., & Seeley, J. R. (2003). The symptomatic expression of major depressive disorder in adolescents and young adults. *Journal of Abnormal Psychology*, 112, 244–252.
- Lewinsohn, P. M., Rohde, P., Seeley, J. R., Klein, D. N., & Gotlib, I. H. (2000). Natural course of adolescent major depressive disorder in a community sample: Predictors of recurrence in young adults. *American Journal of Psychiatry*, 157, 1584–1591.
- Lichenstein, P., & Annas, P. (2000). Heritability and prevalence of specific fears and phobias in childhood. *Journal of Child Psychology and Psychiatry and Allied Disciplines*, 41, 927–937.
- Lichtenstein, P., Carlstrom, E., Ramstam, M., Gillberg, C., & Anckarsater, H. (2010). The genetics of autism spectrum disorders and related neuropsychiatric disorders in childhood. *American Journal of Psychiatry*, 167, 1357–1363.
- Linnet, K. M., Dalsgaard, S., Obel, C., et al. (2003). Maternal lifestyle factors in pregnancy risk of attention deficit hyperactivity disorder and associated behaviors: Review of the current evidence. *American Journal of Psychiatry*, 160, 1028–1040.
- Loeber, R., Burke, J. D., Lahey, B. B., Winters, A., & Zera, M. (2000). Oppositional defiant and conduct disorder: A review of the past 10 years, Part I. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 1468–1484.
- Loeber, R., & Keenan, K. (1994). Interaction between conduct disorder and its comorbid conditions: Effects of age and gender. *Clinical Psychology Review*, 14, 497–523.
- Lord, C., Risi, S., DiLavore, P. S., Shulman, C., Thurm, A., & Pickles, A. (2006). Autism from 2 to 9 years of age. *Archives of General Psychiatry*, 63, 694–701.
- Lovaas, O. I. (1987). Behavioral treatment and normal educational and intellectual functioning in young autistic children. *Journal of Consulting and Clinical Psychology*, 55, 3–9.
- Lynam, D., & Henry, B. (2001). The role of neuropsychological deficits in conduct disorders. In J. Hill & B. Maughan (Eds.), *Conduct disorders in childhood and adolescence* (pp. 235–263). New York: Cambridge University Press.
- Lynam, D., Moffitt, T. E., & Stouthamer-Loeber, M. (1993). Explaining the relation between IQ and delinquency: Race, class, test motivation, school failure, or self-control. *Journal of Abnormal Psychology*, 102, 187–196.
- MacDonald, V. M., Tsiantis, J., Achenbach, T. M., Motti-Stefanidi, F., & Richardson, C. (1995). Competencies and problems reported by parents of Greek and American children, ages 6–11. *European Child and Adolescent Psychiatry*, 4, 1–13.
- Mann, V. A., & Brady, S. (1988). Reading disability: The role of language deficiencies. *Journal of Consulting and Clinical Psychology*, 56, 811–816.
- Mannuzza, S., Klein, R. G., Bonagura, N., Malloy, P., Giampino, T. L., & Addalli, K. A. (1991). Hyperactive boys almost grown up. V. Replication of psychiatric status. *Archives of General Psychiatry*, 48(1), 77–83.
- Mannuzza, S., Klein, R. G., & Moulton, J. L. (2003). Does stimulant medication place children at risk for adult substance abuse? A controlled, prospective follow-up study. *Journal of Child and Adolescent Psychopharmacology*, 13, 273–282.
- March, J., Silva, S., Petrycki, S., et al. (2004). Fluoxetine, cognitive-behavioral therapy, and their combination for adolescents with depression: Treatment for adolescents with depression study (TADS) randomized controlled trial. *Journal of the American Medical Association*, 292, 807–820.
- Marsh, A. A., & Blair, R. J. (2008). Deficits in facial affect recognition among antisocial populations: A meta-analysis. *Neuroscience and Biobehavioral Reviews*, 32(3), 454–465.
- Martini, D. R., Ryan, C., Nakayama, D., & Ramenofsky, M. (1990). Psychiatric sequelae after traumatic injury: The Pittsburgh regatta accident. *Journal of the American Academy of Child and Adolescent Psychiatry*, 29, 70–75.
- McClellan, J., Kowatch, R., & Findling, R. L. (2007). Practice parameter for the assessment and treatment of children and adolescents with bipolar disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 107–125.

- McEachin, J. J., Smith, T., & Lovaas, O. I. (1993). Longterm outcome for children with autism who received early intensive behavioral treatment. *American Journal on Mental Retardation*, 97, 359–372.
- McGovern, C. W., & Sigman, M. (2005). Continuity and change from early childhood to adolescence in autism. *Journal of Child Psychology and Psychiatry*, 46, 401–408.
- McLeod, B. D., Weisz, J. R., & Wood, J. J. (2007). Examining the association between parenting and childhood: A meta-analysis. *Clinical Psychology Review*, 27, 986–1003.
- McLeod, B. D., Wood, J. J., & Weisz, J. R. (2007). Examining the association between parenting and childhood anxiety: A meta-analysis. *Clinical Psychology Review*, 27, 155–172.
- Merikangas, K. R., He, J. P., Brody, D., Fisher, P. W., Bourdon, K., & Koretz, D. S. (2010). Prevalence and treatment of mental disorders among US children in the 2001–2004 NHANES. *Pediatrics*, 125(1), 75–81.
- Mikami, A. Y., Hinshaw, S. P., Arnold, L. E., Hoza, B., Hechtman, L., Newcorn, J. H., & Abikoff, H. B. (2010). Bulimia nervosa symptoms in the multimodal treatment study of children with ADHD. *International Journal of Eating Disorders*, 43, 248–259.
- Mikami, A. Y., Huang-Pollack, C. L., Piffner, L. J., McBurnett, K., & Hangai, D. (2007). Social skills differences among attention-deficit/hyperactivity disorder types in a chat room assessment task. *Journal of Abnormal Child Psychology*, 35, 509–521.
- Moffitt, T. E. (1993). Adolescence-limited and life-course-persistent antisocial behavior: A developmental taxonomy. *Psychological Review*, 100, 674–701.
- Moffitt, T. E., & Caspi, A. (2001). Childhood predictors differentiate life-course persistent and adolescence-limited antisocial pathways among males and females. *Developmental Psychopathology*, 13(2), 355–375.
- Moffitt, T. E. (2007). A review of research on the taxonomy of life-course persistent versus adolescence-limited antisocial behavior. In D. J. Flannery, A. T. Vazsonyi, & I. D. Waldman (Eds.), *The Cambridge handbook of violent behavior and aggression* (pp. 49–74). New York: Cambridge University Press.
- Moffitt, T. E., Caspi, A., Harrington, H., & Milne, B. J. (2002). Males on the life-course persistent and adolescence-limited antisocial pathways: Follow-up at age 26. *Development and Psychopathology* 14, 179–207.
- Moffitt, T. E., Lynam, D., & Silva, P. A. (1994). Neuropsychological tests predict persistent male delinquency. *Criminology*, 32, 101–124.
- Moffitt, T. E., & Silva, P. A. (1988). IQ and delinquency: A direct test of the differential detection hypothesis. *Journal of Abnormal Psychology*, 97, 330–333.
- Molina, B. S. G., Hinshaw, S. P., Swanson, J. M., Arnold, L. E., Vitiello, B., Jensen, P. S., et al. (2009). The MTA at 8 years: Prospective follow-up of children treated for combined-type ADHD in a multisite study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 48, 484–500.
- Molina, B. S., Hinshaw, S. P., Eugene Arnold, L., Swanson, J. M., Pelham, W. E., Hechtman, L., et al. (2013). Adolescent substance use in the multimodal treatment study of attention-deficit/hyperactivity disorder (ADHD) (MTA) as a function of childhood ADHD, random assignment to childhood treatments, and subsequent medication. *Journal of the American Academy of Child and Adolescent Psychiatry*, 52(3), 250–263.
- Monuteaux, M., Faraone, S. V., Gross, L., & Biederman, J. (2007). Predictors, clinical characteristics, and outcome of conduct disorder in girls with attention-deficit/hyperactivity disorder: a longitudinal study. *Psychological Medicine*, 37, 1731–1741.
- MTA Cooperative Group. (1999a). A 14-month randomized clinical trial of treatment strategies for attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, 56, 1073–1086.
- MTA Cooperative Group. (1999b). Moderators and mediators of treatment response for children with attention-deficit/hyperactivity disorder. *Archives of General Psychiatry*, 56, 1088–1096.
- Munson, J., Dawson, H., Abbott, R., Faja, S., Webb, S. J., Friedman, S. D., . . . Dager, S. (2006). Amygdalar volume and behavioral development in autism. *Archives of General Psychiatry*, 63(6), 686–693.
- Murray-Close, D., Hoza, B., Hinshaw, S. P., Arnold, L. E., Swanson, J., Jensen, P. S., Hechtman, L., & Wells, K. (2010). Developmental processes in peer problems of children with attention-deficit/hyperactivity disorder in The Multimodal Treatment Study of Children with ADHD: Developmental cascades and vicious cycles. *Developmental and Psychopathology*, 22, 785–802.
- Nacewicz, B. M., Dalton, K. M., Johnstone, T. et al. (2006). Amygdala volume and nonverbal social impairment in adolescent and adult males with autism. *Archives of General Psychiatry*, 63, 1417–1448.
- National Fragile X Foundation. (n.d.). *Fmr1 gene*. Retrieved from <https://fragilex.org/fragile-x/genetics-and-inheritance/fmr1-gene/>
- National Institute of Child Health and Human Development. (2000). Report of the National Reading Panel. Teaching children to read: An evidence-based assessment of the scientific research literature on reading and its implications for reading instruction. Retrieved from [www.nichd.nih.gov/publications/nrp/smallbook.htm](http://www.nichd.nih.gov/publications/nrp/smallbook.htm).
- Naylor, B. T. (2009). *Depression — causes, diagnosis and treatment: Depression in children*. Hauppauge, US: Nova Science Publishers.
- Neuman, R. J., Lobos, E., Reich, W., Henderson, C. A., Sun, L. W., & Todd, R. D. (2007). Prenatal smoking exposure and dopaminergic genotypes interact to cause a severe ADHD subtype. *Biological Psychiatry*, 61, 1320–1328.
- Nigg, J. T. (2013). Attention deficits and hyperactivity-impulsivity: What have we learned, what next? *Development and Psychopathology*, 25(4 Pt 2), 1489–1503.

- Nigg, J. T., & Casey, B. J. (2005). An integrative theory of attention-deficit/hyperactivity disorder based on the cognitive and affective neurosciences. *Development and Psychopathology*, 17, 785–806.
- Nigg, J. T., Lewis, K., Edinger, T., & Falk, M. (2012). Meta-analysis of attention-deficit/hyperactivity disorder or attention-deficit/hyperactivity disorder symptoms, restriction diet, and synthetic food color additives. *Journal of the American Academy of Child and Adolescent Psychiatry*, 51(1), 86–97.e88.
- Nock, M. K., Kazdin, A. E., Hiripi, E., & Kessler, R. C. (2006). Prevalence, subtypes, and correlates of DSM-IV conduct disorder in the National Comorbidity Survey Replication. *Psychological Medicine*, 36, 699–710.
- O’Kearney, R. T., Anstey, K. J., & von Sanden, C. (2006). Behavioural and cognitive behavioural therapy for obsessive compulsive disorder in children and adolescents. *Cochrane Database of Systematic Reviews* 2006, Issue 4.
- Odgers, C. L., Caspi, A., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., Poulton, R., Sears, M. R., Thomson, W. M., & Moffitt, T. E. (2007). Prediction of differential adult health burden by conduct problem subtypes in males. *Archives of General Psychiatry*, 64, 476–484.
- Odgers, C. L., Moffitt, T. E., Broadbent, J. M., Dickson, N., Hancox, R. J., Harrington, H., Poulton, R., Sears, M. R., Thomson, W. M., & Caspi, A. (2008). Female and male antisocial trajectories: From childhood origins to adult outcomes. *Developmental and Psychopathology*, 20, 673–716.
- Ogden, T., & Halliday-Boykins, C. A. (2004). Multisystemic treatment of antisocial adolescents in Norway: Replication of clinical outcomes outside the U.S. *Child and Adolescent Mental Health Volume*, 9, 77–83.
- Ortiz, J., & Raine, A. (2004). Heart rate level and antisocial behavior in children and adolescents: A meta-analysis. *Journal of the American Academy of Child and Adolescent Psychiatry*, 43, 154–162.
- Patterson, G. R. (1982). *Coercive family process*. Eugene, OR: Castilia.
- Pediatric OCD treatment study (POTS) team. (2004). Cognitive-behavior therapy, setraline, and their combination for children and adolescents with obsessive-compulsive disorder: The pediatric OCD treatment study (POTS) randomized controlled trial. *Journal of the American Medical Association*, 292, 1969–1976.
- Pelham, W. E., Gnagy, E. M., Greiner, A. R., Hoza, B., Hinshaw, S. P., Swanson, J. M., Simpson, S., Shapiro, C., Bukstein, O., Baron-Myak, C., & McBurnett, K. (2000). Behavioral versus behavioral plus pharmacological treatment for ADHD children attending a summer treatment program. *Journal of Abnormal Child Psychology*, 28, 507–525.
- Pennington, B. F. (1995). Genetics of learning disabilities. *Journal of Child Neurology*, 10, S69–S77.
- Perry, R., Campbell, M., Adams, P., Lynch, N., Spencer, E. K., Curren, E. L., & Overall, J. E. (1989). Longterm efficacy of haloperidol in autistic children: Continuous versus discontinuous administration. *Journal of the American Academy of Child and Adolescent Psychiatry*, 28, 87–92.
- Piacentini, J., Bennett, S., Compton, S. N., Kendall, P. C., Birmaher, B., Albano, A. M., et al. (2014). 24- and 36-week outcomes for the Child/Adolescent Anxiety Multimodal Study (CAMS). *Journal of the American Academy of Child and Adolescent Psychiatry*, 53(3), 297–310.
- Pierce, K., & Courchesne, E. (2001). Evidence for a cerebellar role in reduced exploration and stereotyped behavior in autism. *Biological Psychiatry*, 49, 655–664.
- Pierce, K., Haist, F., Sedaghat, F., & Courchesne, E. (2004). The brain response to personally familiar faces in autism: Findings of fusiform activity and beyond. *Brain*, 127, 1–14.
- Pierce, K., Muller, R. A., Ambrose, J., Allen, G., & Courchesne, E. (2001). Face processing occurs outside the fusiform “face area” in autism: Evidence from functional MRI. *Brain*, 124, 2059–2073.
- Piven, J., Arndt, S., Bailey, J., & Andreasen, N. (1996). Regional brain enlargement in autism: A magnetic resonance imaging study. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 530–536.
- Piven, J., Arndt, S., Bailey, J., Havercamp, S., Andreasen, N. C., & Palmer, P. (1995). An MRI study of brain size in autism. *American Journal of Psychiatry*, 152, 1145–1149.
- Plomin, R., & Kovas, Y. (2005). Generalist genes and learning disabilities. *Psychological Bulletin*, 131, 592–617.
- Polanczyk, G., de Lima, M. S., Horta, B. L., Biederman, J., & Rohde, L. A. (2007). The worldwide prevalence of ADHD: A systematic review and metaregression analysis. *American Journal of Psychiatry*, 164(6), 942–948.
- Posey, M. J., & McDougle, C. M. (2000). The pharmacotherapy of target symptoms associated with autistic disorder and other pervasive developmental disorders. *Harvard Review of Psychiatry*, 8, 45–63.
- Prieto, S. L., Cole, D. A., & Tager-Flusberg, C. W. (1992). Depressive self-schemas in clinic and nonclinic children. *Cognitive Therapy and Research*, 16, 521–534.
- Raine, A., Venables, P. H., & Williams, M. (1990). Relationships between central and autonomic measures of arousal at age 15 years and criminality at age 24 years. *Archives of General Psychiatry*, 47, 1003–1007.
- Ramus, F. (2014). Neuroimaging sheds new light on the phonological deficit in dyslexia. *Trends in Cognitive Sciences*, 18(6), 274–275.
- Rapee, R. M., Abbott, M., & Lyneham, H. (2006). Bibliotherapy for children with anxiety disorders using written materials for parents: A randomized controlled trial. *Journal of Consulting and Clinical Psychology*, 74, 436–444.
- Rapee, R. M., Schniering, C. A., & Hudson, J. L. (2009). Anxiety disorders during childhood adolescence: Origins and treatment. *Annual Review of Clinical Psychology*, 5, 311–341.
- Raskind, W. H. (2001). Current understanding of the genetic basis of reading and spelling disability. *Learning Disability Quarterly*, 24, 141–157.

- Raznahan, A., Wallace, G. L., Antezana, L., Greenstein, D., Lenroot, R., Thurm, A., et al. (2013). Compared to what? Early brain overgrowth in autism and the perils of population norms. *Biological Psychiatry*, 74(8), 563–575.
- Regier, D. A., Narrow, W. E., Clarke, D. E., Kraemer, H. C., Kuramoto, S. J., Kuhl, E. A., & Kupfer, D. J. (2013). DSM-5 field trials in the United States and Canada, Part II: Test-retest reliability of selected categorical diagnoses. *American Journal of Psychiatry*, 170, 59–70.
- Reid, D. H., Wilson, P. G., & Faw, G. D. (1991). Teaching self-help skills. In J. L. Matson & J. A. Mulick (Eds.), *Handbook of Mental Retardation*. New York: Pergamon Press.
- Reynolds, S., Wilson, C., Austin, J., & Hooper, L. (2012). Effects of psychotherapy for anxiety in children and adolescents: A meta-analytic review. *Clinical Psychology Review*, 32(4), 251–262.
- Rhee, S. H., & Waldman, I. D. (2002). Genetic and environmental influences on antisocial behavior: A meta-analysis of twin and adoption studies. *Psychological Bulletin*, 128, 490–529.
- Rhode, P., Seeley, J. R., Kaufman, N. K., Clarke, G. N., & Stice, E. (2006). Predicting time to recovery among depressed adolescents treated in two psychosocial group interventions. *Journal of Consulting and Clinical Psychology*, 74, 80–88.
- Ricks, D. M. (1972). *The beginning of vocal communication in infants and autistic children*. Doctoral dissertation, University of London.
- Rimland, B. (1964). *Infantile autism*. New York: Appleton-Century-Crofts.
- Rolon-Arroyo, B., Arnold, D. H., & Harvey, E. A. (2013). The predictive utility of conduct disorder symptoms in preschool children: A 3-year follow-up study. *Child Psychiatry and Human Development*.
- Rutter, M., Caspi, A., Fergusson, D., Horwood, L. J., Goodman, R., Maughan, B., Moffitt, T. E., Meltzer, H., & Carroll, J. (2004). Sex differences in developmental reading disability: New findings from 4 epidemiological studies. *Journal of the American Medical Association*, 291, 2007–2012.
- Sacks, O. (1995). *An anthropologist on Mars*. New York: Knopf.
- Scarborough, H. S. (1990). Very early language deficits in dyslexic children. *Child Development*, 61, 128–174.
- Schalock, R. L., Luckasson, R. A., & Shogren, K. A., et al. (2007). The renaming of mental retardation: Understanding the change to the term intellectual disability. *Intellectual and Developmental Disabilities*, 45, 116–124.
- Schecter, R., & Grether, J. K. (2008). Continuing increases in autism reported to California's Developmental Services System. *Archives of General Psychiatry*, 65, 19–24.
- Schnab, D. W., & Trinh, N. G. (2004). Do artificial food colors promote hyperactivity in children with hyperactive syndromes? A meta-analysis of double-blind placebo-controlled trials. *Journal of Developmental and Behavioral Pediatrics*, 25, 425–434.
- Schopler, E., Short, A., & Mesibov, G. (1989). Relation of behavioral treatment to "normal functioning": Comment on Lovaas. *Journal of Consulting and Clinical Psychology*, 57, 162–164.
- Shaw, D. S., Dishion, T. J., Supplee, L., Gardner, F., & Arnds, K. (2006). Randomized trial of a family-centered approach to the prevention of early conduct problems: 2-year effects of the family check-up in early childhood. *Journal of Consulting and Clinical Psychology*, 74, 1–9.
- Shaywitz, B. A., Shaywitz, S. E., Blachman, B. A., et al. (2004). Development of left occipitotemporal systems for skilled reading in children after a phonologically-based intervention. *Biological Psychiatry*, 55, 926–933.
- Shaywitz, B. A., Shaywitz, S. E., Pugh, K. R., et al. (2002). Disruption of posterior brain systems for reading in children with developmental dyslexia. *Biological Psychiatry*, 52, 101–110.
- Sherman, D. K., Iacono, W. G., & McGue, M. K. (1997). Attention-deficit hyperactivity disorder dimensions: A twin study of inattention and impulsivity-hyperactivity. *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 745–753.
- Shic, F., Macari, S., & Chawarska, K. (2014). Speech disturbs face scanning in 6-month-old infants who develop autism spectrum disorder. *Biological Psychiatry*, 75(3), 231–237.
- Shin, M., Besser, L. M., Kucik, J. E., Lu, C., Siffel, C., & Correa, A. (2009). Prevalence of Down syndrome among children and adolescents in 10 regions of the United States. *Pediatrics*, 124, 1565–1571.
- Sigman, M. (1994). What are the core deficits in autism? In S. H. Broman & J. Grafman (Eds.), *Atypical cognitive deficits in developmental disorders: Implications for brain function* (pp. 139–157). Hillsdale, NJ: Erlbaum.
- Simonoff, E. (2001). Genetic influences on conduct disorder. In J. Hill & B. Maughan (Eds.), *Conduct disorders in childhood and adolescence* (pp. 202–234). Cambridge, UK: Cambridge University Press.
- Siok, W. T., Perfetti, C. A., Lin, Z., & Tan, L. H. (2004). Biological abnormality of impaired reading is constrained by culture. *Nature*, 431, 71–76.
- Smith, T., Groen, A., & Wynn, J. W. (2000). Randomized trial of intensive early intervention for children with pervasive developmental disorder. *Research in Developmental Disabilities*, 21, 297–309.
- Spencer, T., Biederman, J., Wilens, T., Harding, M., O'Donnell, D., & Griffin, S. (1996). Pharmacotherapy of attention-deficit hyperactivity disorder across the life cycle. *Journal of the American Academy of Child and Adolescent Psychiatry*, 35, 409–432.
- Sprich, S., Biederman, J., Crawford, M. H., Mundy, E., & Faraone, S. V. (2000). Adoptive and biological families of children and adolescents with ADHD. *Journal of the American Academy of Child and Adolescent Psychiatry*, 39, 1432–1437.
- St. Pourcain, B., Wang, K., Glessner, J. T., Golding, J., Steer, C., Ring, S. M., Skuse, D. H., Grant, S. F. A., Hakonarson, H., & Smith, G. D. (2010). Association between a high-risk autism locus on 5p14 and social communication spectrum phenotypes in the general population. *American Journal of Psychiatry*, 167, 1364–1372.

- Stanfield, A. C., McIntosh, A. M., Spencer, M. D., Philip, R., Gaur, S., & Lawrie, S. M. (2008). Towards a neuroanatomy of autism: A systematic review and meta-analysis of structural magnetic resonance imaging studies. *European Psychiatry*, 23(4), 289–299.
- Steinhausen, H. C., & Metzke, C. W. (1998). Youth self-report of behavioral and emotional problems in a Swiss epidemiological study. *Journal of Youth and Adolescence*, 27, 429–441.
- Strong, G. K., Torgerson, C. J., Torgerson, D., & Hulme, C. (2011). A systematic meta-analytic review of evidence for the effectiveness of the “Fast ForWord” language intervention program. *Journal of Child Psychology and Psychiatry*, 52(3), 224–235.
- Sullivan, P. F., Daly, M. J., & O’Donovan, M. (2012). Genetic architectures of psychiatric disorders: The emerging picture and its implications. *Nature Reviews Genetics*, 13(8), 537–551.
- Swanson, J., Hinshaw, S. P., Arnold, L. E., Gibbons, R., Marcus, S., Hur, K., et al. (2007). Secondary evaluations of MTA 36-month outcomes: Propensity score and growth mixture model analyses. *Journal of the American Academy of Child and Adolescent Psychiatry*, 46, 1002–1013.
- Swanson, J., Kinsbourne, M., Nigg, J., et al. (2007). Etiologic subtypes of attention-deficit/hyperactivity disorder: Brain imaging, molecular genetic and environmental factors and the dopamine hypothesis. *Neuropsychology Review*, 17, 39–59.
- Swanson, J., McBurnett, K., Christian, D. L., & Wigal, T. (1995). Stimulant medications and the treatment of children with ADHD. In T. H. Ollendick & R. J. Prinz (Eds.), *Advances in Clinical Child Psychology* (Vol. 17, pp. 265–322). New York: Plenum.
- TADS team. (2007). The treatment for adolescents with depression study (TADS): Long term effectiveness and safety outcomes. *Archives of General Psychiatry*, 64, 1132–1144.
- Tallal, P., Miller, S. L., Bedi, G., Byma, G., Wang, X., Nagarajan, S. S., Schreiner, C., Jenkins, W. M., & Merzenich, M. M. (1996). Language comprehension in languagelearning impaired children improved with acoustically modified speech. *Science*, 271, 81–84.
- Tallmadge, J., & Barkley, R. A. (1983). The interactions of hyperactive and normal boys with their mothers and fathers. *Journal of Abnormal Child Psychology*, 11, 565–579.
- Taylor, A., & Kim-Cohen, J. (2007). Meta-analysis of gene–environment interactions in developmental psychopathology. *Development and Psychopathology*, 19, 1029–1037.
- Taylor, J., Iacono, W. G., & McGue, M. (2000). Evidence for a genetic etiology of early-onset delinquency. *Journal of Abnormal Psychology*, 109, 634–643.
- Teachman, B. A., & Allen, J. P. (2007). Development of social anxiety: Social interaction predictors of implicit and explicit fear of negative evaluation. *Journal of Abnormal Child Psychology*, 35, 63–78.
- Thapar, A., Langley, K., Owen, M. J., & O’Donovan, M. C. (2007). Advances in genetic findings on attention deficit hyperactivity disorder. *Psychological Medicine*, 37, 1681–1692.
- Thapar, A., Rice, F., Hay, D., Boivin, J., Langley, K., van den Bree, M., et al. (2009). Prenatal smoking might not cause attention-deficit/hyperactivity disorder: evidence from a novel design. *Biological Psychiatry*, 66(8), 722–727.
- Tully, L. A., Arseneault, L., Caspi, A., Moffitt, T. E., & Morgan, J. (2004). Does maternal warmth moderate the effects of birth weight on twins’ attention-deficit/hyperactivity disorder (ADHD) symptoms and low IQ? *Journal of Consulting and Clinical Psychology*, 72, 218–226.
- Turner, C. M. (2006). Cognitive-behavioural theory and therapy for obsessive-compulsive disorder in children and adolescents: Current status and future directions. *Clinical Psychology Review*, 26, 912–948.
- Van Meter, A. R., Moreira, A. L. R., & Youngstrom, E. A. (2011). Meta-analysis of epidemiologic studies of pediatric bipolar disorder. *Journal of Clinical Psychiatry*, 72(9), 1250–1256.
- Vandermosten, M., Boets, B., Poelmans, H., Sunaert, S., Wouters, J., & Ghesquiere, P. (2012). A tractography study in dyslexia: neuroanatomic correlates of orthographic, phonological and speech processing. *Brain*, 135(Pt 3), 935–948.
- Victorian State Government. (2015). The Victorian public health and wellbeing plan 2015–2019. Retrieved from [www2.health.vic.gov.au/about/health-strategies/public-health-wellbeing-plan](http://www2.health.vic.gov.au/about/health-strategies/public-health-wellbeing-plan)
- Virués-Ortega, J. (2010). Applied behavior analytic intervention for autism in early childhood: Meta-analysis, meta-regression and dose–response meta-analysis of multiple outcomes. *Clinical Psychology Review*, 30, 387–399.
- Volkow, N. D., Wang, G. J., Kollins, S. H., Wigal, T. L., Newcorn, J. H., Telang, F., et al. (2009). Evaluating dopamine reward pathway in ADHD: Clinical implications. *Journal of the American Medical Association*, 302(10), 1084–1091.
- Volkow, N. D., Wang, G. J., Newcorn, J. H., Kollins, S. H., Wigal, T. L., Telang, F., et al. (2011). Motivation deficit in ADHD is associated with dysfunction of the dopamine reward pathway. *Molecular Psychiatry*, 16(11), 1147–1154.
- Vrshek-Schallhorn, S., Doane, L. D., Mineka, S., Zinbarg, R. E., Craske, M. G., & Adam, E. K. (2013). The cortisol awakening response predicts major depression: Predictive stability over a 4-year follow-up and effect of depression history. *Psychological Medicine*, 43(3), 483–493.
- Vrshek-Schallhorn, S., Mineka, S., Zinbarg, R. E., Craske, M. G., Griffith, J. W., Sutton, J., et al. (2013). Refining the candidate environment: Interpersonal stress, the serotonin transporter polymorphism, and gene–environment interactions in major depression. *Clinical Psychological Science*, 2(3), 235–248.
- Wakefield, A. J., Murch, S. H., Anthony, A., Linnell, J., Casson, D. M., Malik, M., . . . Walker-Smith, J. A. (1998). Ileal-lymphoid-nodular hyperplasia, non-specific colitis, and pervasive developmental disorder in children. *The Lancet*, 351(9103), 637–641. Retrieved from [www.thelancet.com/journals/lancet/article/PIIS0140-6736\(97\)11096-0/abstract](http://www.thelancet.com/journals/lancet/article/PIIS0140-6736(97)11096-0/abstract)

- Walkup, J. T., Albano, A. M., Piacentini, J., Birmaher, B., Compton, S. N., Sherrill, J. T., et al. (2008). Cognitive behavioral therapy, sertraline, or a combination in childhood anxiety. *New England Journal of Medicine*, 359, 2753–2766.
- Wang, K., Zhang, H., Ma, D., Bucan, M., Glassner, J. T., Abrahams, B. S., Salyakina, D., et al. (2009). Common genetic variants on 5p14.1 associate with autism spectrum disorders. *Nature*, 459, 528–533.
- Waszczuk, M. A., Zavos, H. M., Gregory, A. M., & Eley, T. C. (2014). The phenotypic and genetic structure of depression and anxiety disorder symptoms in childhood, adolescence, and young adulthood. *Journal of the American Medical Association, Psychiatry*, 71, 905–916.
- Webster-Stratton, C. (1998). Preventing conduct problems in Head Start children: Strengthening parenting competencies. *Journal of Consulting and Clinical Psychology*, 66, 715–730.
- Webster-Stratton, C., Reid, M. J., & Hammond, M. (2001). Preventing conduct problems, promoting social competence: A parent and teacher training partnership in Head Start. *Journal of Clinical Child Psychology*, 30, 283–302.
- Weiss, G., & Hechtman, L. (1993). *Hyperactive children grown up* (2nd ed.). New York: Guilford Press.
- Weiss, L. A., Shen, Y., Korn, J. M., et al. (2008). Association between microdeletion and microduplication at 16p11.2 and autism. *New England Journal of Medicine*, 358, 667–675.
- Weisz, J. R., McCarty, C. A., & Valeri, S. M. (2006). Effects of psychotherapy for depression in children and adolescents: A meta-analysis. *Psychological Bulletin*, 132, 132–149.
- Weisz, J. R., Sigman, M., Weiss, B., & Mosk, J. (1993). Parent reports of behavioral and emotional problems among children in Kenya, Thailand and the United States. *Child Development*, 64, 98–109.
- Weisz, J. R., Suwanlert, S. C., Wanchai, W., & Bernadette, R. (1987). Over- and under-controlled referral problems among children and adolescents from Thailand and the United States: The wat and wai of cultural differences. *Journal of Consulting and Clinical Psychology*, 55, 719–726.
- Weisz, J. R., Weiss, B., Suwanlert, S., & Wanchai, C. (2003). Syndromal structure of psychopathology in children of Thailand and the United States. *Journal of Consulting and Clinical Psychology*, 71, 375–385.
- Wells, K. C., Epstein, J. N., Hinshaw, S. P., et al. (2000). Parenting and family stress treatment outcomes in attention deficit hyperactivity disorder (ADHD): An empirical analysis in the MTA study. *Journal of Abnormal Child Psychology*, 28, 543–553.
- Whitaker, A. H., van Rossen, R., Feldman, J. F., Schonfeld, I. S., Pinto-Martin, J. A., Torre, C., Shaffer, D., & Paneth, N. (1997). Psychiatric outcomes in low birth weight children at age 6 years: Relation to neonatal cranial ultrasound abnormalities. *Archives of General Psychiatry*, 54, 847–856.
- White, S. W., Oswald, D., Ollendick, T., & Scahill, L. (2009). Anxiety in children and adolescents with autism spectrum disorders. *Clinical Psychology Review*, 29, 216–229.
- Whittle, S., Lichter, R., Dennison, M., Vijayakumar, N., Schwartz, O., Byrne, M. L., et al. (2014). Structural brain development and depression onset during adolescence: A prospective longitudinal study. *The American Journal of Psychiatry*, 171(5), 564–571.
- Willemsen-Swinkels, S. H. N., Buitelaar, J. K., Weijnen, F. G., & van Engeland, H. (1995). Placebo-controlled acute dosage naltrexone study in young autistic children. *Psychiatry Research*, 58, 203–215.
- Williams, P. A., Allard, A., Spears, L., Dalrymple, N., & Bloom, A. S. (2001). Brief report: Case reports on naltrexone use in children with autism: Controlled observations regarding benefits and practical issues in medication management. *Journal of Autism and Developmental Disorders*, 31, 103–108.
- Wilson, A. J., & Dehaene, S. (2007). Number sense and developmental dyscalculia. In D. Coch (Ed.), *Human behavior, learning and the developing brain: Atypical development* (pp. 212–238). New York: Guilford Press.
- Wolf, M., Bally, H., & Morris, R. (1986). Automaticity, retrieval processes, and reading: A longitudinal study in average and impaired readers. *Child Development*, 57, 988–1000.
- Wolraich, M. L., Wilson, D. B., & White, J. W. (1995). The effect of sugar on behavior or cognition in children: A meta-analysis. *Journal of the American Medical Association*, 274, 1617–1621.
- Yirmiya, N., & Sigman, M. (1991). High functioning individuals with autism: Diagnosis, empirical findings, and theoretical issues. *Clinical Psychology Review*, 11, 669–683.
- Youngstrom, E. A., Freeman, A. J., & Jenkins, M. M. (2009). The assessment of children and adolescents with bipolar disorder. *Child and Adolescent Psychiatric Clinics of North America*, 18, 353–390. doi:10.1016/j.chc.2008.1012.1002.

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## CHAPTER 11

# Late life and neurocognitive disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 11.1** differentiate common misconceptions from established findings about age-related changes and discuss issues involved in conducting research on ageing
  - 11.2** describe the prevalence of psychological disorders in the elderly and issues involved in estimating the prevalence
  - 11.3** explain the symptoms, aetiology and treatment of cognitive disorders in the elderly.
-

## OPENING SCENARIO

Joan is a 74-year-old retired school teacher who lives alone. She was brought to her general practitioner (GP) by her son, who had noticed a sudden increase in confusion over the past week. When questioned by the GP, Joan reported that she was doing well and denied any problems. In contrast, her son reported that his mother had not contacted him in over a week when she usually calls daily. Three days earlier he visited Joan and found her to be unusually quiet and confused about where she was. He also found the house uncharacteristically dirty with a slight odour of urine. Joan's medical history includes hypertension, diabetes, insomnia, anxiety, chronic headaches and back pain. Medications include Ramipril, Metformin, Alprazolam and Diazepam. Joan smokes a pack of cigarettes and drinks two to three glasses of wine per day. Approximately one year ago, her son started managing her finances. There is a family history of memory problems.

On examination, Joan was distractible and minimally reactive. She denied feeling sad and reported no delusions or hallucinations; although she did note that her eyes 'play tricks' on her and she sees faces on the bedroom curtain. She was disorientated to time and location, and demonstrated difficulties with recall and attention.

The GP ordered a series of blood and urine tests the same day. Based on the findings and clinical presentation, the GP concluded that Joan's symptoms are the result of a delirium caused by a urinary tract infection (UTI) and exacerbated by dehydration and hyperglycaemia. He prescribed intravenous saline, antibiotics and haloperidol. Joan is monitored over time and, at the follow-up visit a few days later, Joan's mental state had returned to baseline (Lyketsos & Liang, 1999).

### QUESTIONS

1. What are possible differential diagnoses the GP should consider?
2. What are the main features that support a diagnosis of delirium?

## Introduction

In this chapter, we focus on psychological and neurocognitive disorders in late life. We begin by reviewing the importance of understanding late life and late life disorders. We describe common myths about ageing, challenges faced by the elderly and some remarkable strengths that come with growing older. Conducting research on psychological health and ageing, though, is complicated by a few key methodological issues and we describe some of the ways these issues can influence findings. We discuss evidence that the prevalence of psychological disorders such as depression, anxiety and substance abuse in the elderly is quite low. With this information as a backdrop, we turn to dementia and delirium, the main topics of this chapter.

## 11.1 Ageing: issues and methods

**LEARNING OUTCOME 11.1** Differentiate common misconceptions from established findings about age-related changes and discuss issues involved in conducting research on ageing.

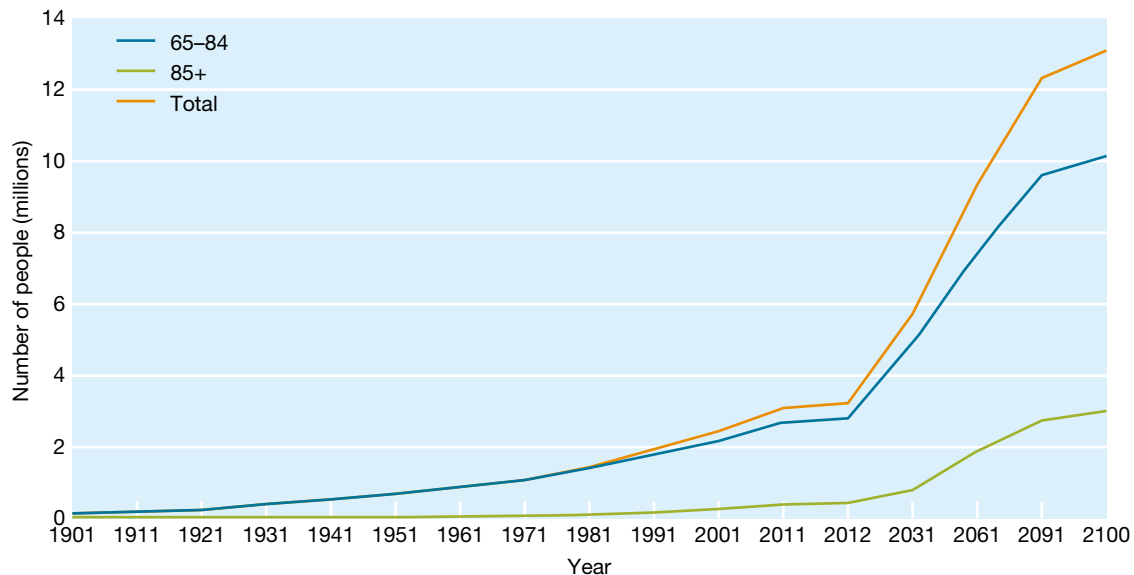
In today's society we live longer and healthier lives than in previous generations. For example, the average life expectancy in Australia in the 1970s was 69.4 years for males and 76.4 years for females; now it is 79.9 for males and 84.3 for females (ABS, 2014b). This represents an average 7.9 to 10.5 year increase in life expectancy over the last 40 years. While many older adults consider themselves in good health, older age is also associated with medical conditions and social change that require special consideration when considering diagnosis and treatment.

Older adults are usually defined as those over the age of 65, an arbitrary point set largely by social policies rather than any physiological process. To have some rough demarcation points, gerontologists usually divide people over age 65 into three groups: the young-old, those aged 65 to 74; the old-old, those aged 75 to 84; and the oldest-old, those over age 85.

People aged over 65 years represent the fastest growing demographic in the majority of countries (United Nations, 2015). At the time of the last census, people 65 and older comprised 14.9 percent (almost 3.5 million) of the Australian population and 14.7 percent (approximately 679 400) of the New Zealand population. Figure 11.1 shows the increase in the number of older adults in Australia over time. In 2012, there were 420 300 people aged over 85 years and this is expected to double by 2031 to 842 500 (ABS, 2014a).

**FIGURE 11.1**

The number of older adults is increasing in Australia. The figure shows the number of people aged over 65 years based on Australian Bureau of Statistics (ABS) Historical Population Statistics from 1901 to 2012 (ABS, 2014b); and projected data from 2012 to 2100 (ABS, 2013). As you can see in the figure, the population of older adults is expected to almost double after 2031.



Given these statistics, it is not surprising that 69 percent of practising psychologists conduct clinical work with older adults (Qualls, Segal, Norman, Niederehe, & Gallagher-Thompson, 2002). A major concern, though, is that fewer than 30 percent of psychologists report receiving any formal training about late-life issues (Qualls et al., 2002) and in a survey of 1498 psychologists Australia-wide, only 4.8 percent reported specialising in working with older adults (Koder & Helmes, 2008).

## Myths about late life

Ethical principles state that it is important for psychologists working with the elderly to examine their attitudes and beliefs about ageing and how this may impact on their practice (American Psychiatric Association, APA, 2004; Australian Psychological Society, APS, 2014; Pachana, Helmes, & Koder, 2006). There are several common myths and assumptions about old age, including the idea that we will become doddering and confused. We worry that we will be unhappy, cope poorly with troubles, become focused on our poor health and lead a lonely life.

Each of these myths has been debunked. As we will see, severe cognitive problems do not occur for most people in late life, though a mild decline in cognitive functioning is common (Langa et al., 2008). Older adults actually experience less negative emotion than do young people (Charles, Reynolds, & Gatz, 2001; Mather & Carstensen, 2005). Although some might suspect that these findings are artefacts of a reluctance of older individuals to describe negative feelings to researchers, laboratory studies verify that the elderly are actually more skilled at regulating their emotions. For example, when shown positive and negative images, older people tend to pay more attention to the positive images (Isaacowitz, 2012) and to display less psychophysiological response to the negative images than do younger people (Kisley,

Wood, & Burrows, 2007; Levenson, Carstensen, & Gottman, 1994). When viewing positive images, they show more robust brain activation in key emotion regions than do younger people (Mather et al., 2004). Negative mood has also been found to persist for shorter periods of time in older adults, suggesting that older adults are better able to dissipate negative affect than younger adults (Carstensen, Pasupathi, Mayr, & Nesselroade, 2000). Many older people under-report somatic symptoms, perhaps because of beliefs that aches and pains are an inevitable part of late life. People in late life are no more likely to meet criteria for somatic symptom disorders than are the young (Regier, Boyd, Burke, & Rae, 1988; Siegler & Costa, 1985).

Another myth, that older people are lonely, has received considerable attention. The truth is that the number of social activities that older people engage in is unrelated to their psychological wellbeing (Carstensen, 1996). As we age, our interests shift away from seeking new social interactions to cultivating a few social relationships that really matter to us, such as those with family and close friends. This phenomenon has been called **social selectivity**.

When we have less time ahead of us, we tend to place a higher value on emotional intimacy than on exploring the world. This preference applies not just to older people but also to younger people who see themselves as having limited time, such as those who are preparing to move far away from their home or who have a life-threatening illness (Frederickson & Carstensen, 1990). When we cannot see a future without end, we prefer to spend our limited time with our closest ties rather than with casual acquaintances. Older people do not tend to report that they want more social contacts (York Cornwell & Waite, 2009). To those unfamiliar with these age-related changes, social selectivity could be misinterpreted as harmful social withdrawal. This body of research highlights some of the many strengths that come with ageing.

Many stereotypes we hold about the elderly are false, but considerable research suggests that the negative attitudes about the elderly learned early in life persist and become negative self-perceptions as people move into their later years (Levy, 2003). These negative self-perceptions have serious consequences. For example, in one experiment, researchers randomly assigned elderly people to a condition in which they were reminded of negative stereotypes of ageing (like memory loss or slower walking speed) or to a control condition without such reminders (Hausdorff, Levy, & Wei, 1999). Participants who were reminded of the negative stereotypes did worse on a memory test than did the control group (Levy, 1996). In a second experimental study, reminders of *positive* stereotypes about ageing led participants to walk more quickly compared to a control condition with no reminders (Hausdorff et al., 1999). Of much more concern than how stereotypes might influence memory and walking speed, researchers have also shown that negative self-views about ageing can predict earlier death, even controlling for baseline health status and many other potential confounds (Levy, Slade, & Kasl, 2002). Not only do we need to challenge our own negative stereotypes, but we also need to help older adults challenge those views.

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As illustrated by the late John Glenn's space flight at age 77, advancing age need not lead to a curtailment of activities.



## The problems experienced in late life

We know that mental health is tied to the physical and social problems in a person's life. As a group, older adults have more of these experiences. For example, older age is associated with physical decline,

sensory acuity deficits, loss of loved ones, social stress of stigmatising attitudes towards the elderly and the cumulative effects of a lifetime of unfortunate experiences. Eighty percent of elderly people have at least one major medical condition (National Academy on an Aging Society, 1999). As described by one author, 'Late life would qualify as the Olympics of coping' (Fisher, 2011, p. 145).

One facet of ageing deserves particular attention. As people age, the quality and depth of sleep decline (Fetveit, 2009) and as many as 50 percent of older adults report difficulties initiating or maintaining sleep (Crowley, 2011). Rates of sleep apnoea, a disorder in which a person stops breathing for seconds to minutes during the night, also increase with age (Prechter & Shepard, 1990). Untreated and chronic sleep deficits can worsen physical, psychological and cognitive problems and can even increase the risk of mortality (Ancoli et al., 1996). Fortunately, psychological treatment has been shown to reduce insomnia among the elderly (Fetveit, 2009).

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The quality of sleep diminishes as people age.



Several problems complicate medical treatment for elderly people. One of the main difficulties is that the chronic health problems of older people seldom diminish; physicians focused on identifying cures can become frustrated when none are available (Zarit, 1980). Other problems result from multiple chronic conditions often experienced by older adults coupled with the time pressure of the health care system. All too often, doctors do not check to see if the person is taking other medications or seeing other health service providers. **Polypharmacy**, the prescribing of multiple drugs to a person, can result (Alpert & Gatlin, 2015). About one-third of elderly persons are prescribed at least five medications (Qato et al., 2008). This increases the risk of adverse drug reactions such as side effects and toxicity. Often, physicians prescribe more medications to combat the side effects, thus continuing the vicious circle.

Further complicating the picture is the fact that most psychoactive drugs are tested on younger people. Gauging the appropriate dose for the less efficient metabolism of the kidneys and liver of the older person represents a challenge for the medical practitioner — side effects and toxicity are much more likely as people age (Gallo & Lebowitz, 1999). The increased sensitivity to side effects is a particular problem with psychiatric medications. One review of medical charts of more than 750 000 elderly patients found that more than one-fifth had filled a prescription for a medication deemed inappropriate for people over the age of 65 due to serious side effects (Curtis et al., 2004). Therefore, it is important that the primary care physician of elderly people keep track of all prescribed medications taken, discontinue non-essential drugs and prescribe only the minimum dosages needed.

Polypharmacy is all too common in late life.



## Research methods in the study of ageing

Research on ageing requires an understanding of several special issues. Chronological age is not as simple a variable in psychological research as it might seem. Because other factors associated with age may influence findings (e.g., the social, political and cultural views or experience of that generation), we must be cautious when we attribute differences between age groups solely to the effects of ageing. In the field of ageing, as in studies of childhood development, a distinction is made among three kinds of effects (see table 11.1).

- **Age effects** are the consequences of being a certain chronological age.
- **Cohort effects** are the consequences of growing up during a particular time period with its unique challenges and opportunities. For example, experiences like the Great Depression, a world war, or the global financial crisis shape experiences and attitudes. Similarly, the expectations for marriage have changed drastically in the past century, at least in Western societies, from a focus on stability to a focus on happiness and personal fulfilment.
- **Time-of-measurement effects** are confounds that arise because events at a particular point in time can have a specific effect on a variable that is being studied (Schaie & Hertzog, 1982). For example, people tested right after experiencing the 2005 earthquake in Haiti might demonstrate elevated levels of anxiety.

**TABLE 11.1** Age, cohort and time-of-measurement effects

Age effects	Cohort effects	Time-of-measurement effects
The effects of being a certain age; for example, being old enough to retire and receive the Age Pension	The effects of having grown up during a particular time period; for example, frugality may be increased among those who lived through the Great Depression of the 1930s	The effects of testing people at a particular time in history; for example, people became more frank during the 1990s in their responses to surveys about their sexual behaviour, as media discussion of sexuality increased

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Cohort effects refer to the fact that people of the same chronological age may differ considerably depending on when they were born.



Two major research designs are used to assess developmental change: cross-sectional and longitudinal. In cross-sectional studies, the investigator compares different age groups at the same moment in time on the variable of interest. Suppose that in 1995 we took a poll in New Zealand and found that many interviewees over age 80 spoke with a European accent, whereas those in their 40s and 50s did not. Could we conclude that as people grow older, they develop European accents? Hardly! Cross-sectional studies do not examine the same people over time; consequently, they do not provide clear information about how people change as they age.

In longitudinal studies, the researcher periodically retests one group of people using the same measure over a number of years or decades. For example, the Australian Longitudinal Study of Ageing (ALSA) has been running since 1992. Since this time, researchers have been following 2087 men and women to see how social, biomedical, psychological, behavioural and environmental factors can impact on health and wellbeing. In general, longitudinal designs allow us to trace individual patterns of consistency or change over time. Although longitudinal studies offer fundamental advantages, results can be biased by attrition, in which participants drop out of the study due to death, immobility or lack of interest. When people are no longer available for follow-up because of death, this is called **selective mortality**. Selective mortality results in a particular form of bias, in that results obtained with the remaining sample are more relevant to drawing conclusions about relatively healthy people than about unhealthy people. Beyond attrition due to death, people with the most problems are likely to drop out from a study, whereas the people who remain are usually healthier than the general population. Later in this chapter, we will discuss how these issues of cohort effects and selective mortality might influence estimates of the prevalence of psychological disorder (Kiecolt-Glaser & Glaser, 2002).

## 11.2 Psychological disorders in late life

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**LEARNING OUTCOME 11.2** Describe the prevalence of psychological disorders in the elderly and issues involved in estimating the prevalence.

The DSM criteria are the same for older and younger adults. The process of diagnosis, however, must be considered with care. DSM criteria specify that a psychological disorder should not be diagnosed if the symptoms can be accounted for by a medical condition or medication side effects. Because medical conditions are more common in the elderly, it is particularly important to rule out such explanations. Medical problems such as thyroid problems, Addison's disease, Cushing's disease, Parkinson's disease, Alzheimer's disease, hypoglycaemia, anaemia, testosterone deficits and vitamin deficiencies can produce symptoms that mimic schizophrenia, depression or anxiety. Medical problems can also worsen the course of depression. Angina, congestive heart failure and excessive caffeine consumption may all cause a faster heart rate, which can be mistaken as a symptom of anxiety (Fisher & Noll, 1996). Age-related deterioration in the vestibular system (inner-ear control of one's sense of balance) can account for panic symptoms such as severe dizziness. Depression is also common after strokes or heart attacks (Teper & O'Brien, 2008). Antihypertensive medication, corticosteroids and antiparkinson medications may contribute to depression or anxiety. Clinicians must be extremely careful to consider the interactions between physical and psychological health. With this consideration in mind, we next examine how common it is for older people to have psychological disorders.

### Estimating the prevalence of psychological disorders in late life

The prevalence estimates for psychological disorders defy stereotypes of unhappiness and anxiety in late life. Findings indicate that persons over age 65 have the lowest prevalence of psychological disorders of all age groups. Table 11.2 provides 12-month estimates from the Te Rau Hinengaro: The New Zealand Mental Health Survey, which involved a community sample of 12 992 individuals who completed extensive diagnostic interviews for mental disorders (Oakley Browne, Wells, & Scott, 2006). As shown, every

single disorder was less common in the elderly than in younger adults. Similar results have been found in Australia (ABS, 2007) (see figure 11.2) and the United States (Gum, King-Kallimanis, & Kohn, 2009). Rates of schizophrenia are also low among the elderly (Howard, Rabins, Seeman, & Jeste, 2000) and older adults are less likely to meet criteria for personality disorders compared to younger individuals (Balsis, Gleason, Woods, & Oltmanns, 2007).

**TABLE 11.2** One-year prevalence estimates (%) for psychological disorders by age group

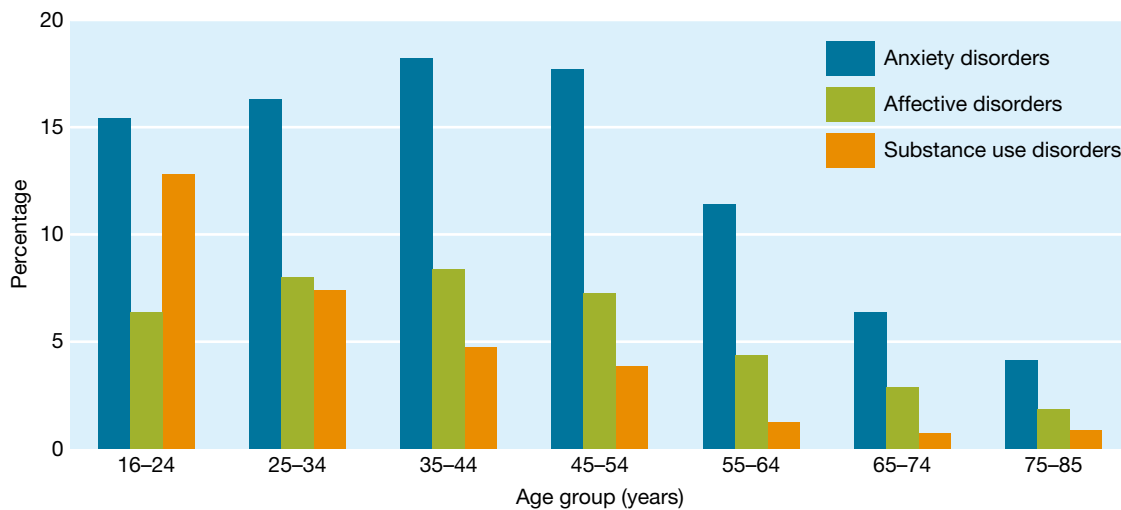
	16–24 years	25–44 years	60–64 years	65 years and older
<b>Anxiety disorders</b>				
Panic disorder	2.4	2.1	1.2	0.6
Agoraphobia without panic	0.7	0.8	0.6	0.2
Specific phobia	9.3	8.3	6.9	3.2
Social phobia	7.0	6.3	4.2	1.4
Generalised anxiety disorder	1.6	2.8	1.8	1.0
Posttraumatic stress disorder <sup>a</sup>	2.4	3.5	3.2	1.7
Obsessive compulsive disorder <sup>a</sup>	1.5	0.8	0.2	0.1
Any anxiety disorder <sup>a</sup>	17.7	18.2	13.2	6.0
<b>Mood disorders</b>				
Major depressive disorder	8.7	6.3	5.2	1.7
Dysthymia	1.5	1.2	1.2	0.4
Bipolar disorder	3.9	2.8	1.4	0.2
Any mood disorder	12.7	9.2	6.8	2.0
<b>Substance disorders<sup>a</sup></b>				
Alcohol abuse	7.1	3.2	0.8	<0.1
Drug abuse	3.8	1.2	0.2	<0.1
Any substance disorder	9.6	4.2	1.2	<0.1
<b>Eating disorders</b>				
Anorexia nervosa <sup>a</sup>	<0.1	<.01	<.01	<0.1
Bulimia <sup>a</sup>	0.6	0.7	0.3	0.1
Any eating disorder <sup>a</sup>	0.6	0.7	0.3	0.1
<b>Any disorder<sup>a</sup></b>	<b>28.6</b>	<b>25.1</b>	<b>17.4</b>	<b>7.1</b>

<sup>a</sup>Assessed in subsample that completed the long interview

**Source:** Adapted from Oakley Browne et al., 2006 (Table 3.1, pp. 41–42).

Beyond examining the prevalence rates of disorder, it is important to consider the incidence rates, or how many people are experiencing the onset of a new disorder. Most people who have an episode of a psychological disorder late in life are experiencing a recurrence of a disorder that started earlier in life rather than an initial onset. For example, 97 percent of older adults with generalised anxiety disorder report that their symptoms began before the age of 65 (Alwahhabi, 2003) and more than 90 percent of older adults with major depressive disorder or agoraphobia report that their symptoms began earlier in life (Norton et al., 2006; Ritchie, Norton, Mann, Carriere, & Ancelin, 2013). Late onset is also extremely rare for schizophrenia (Karon & VandenBos, 1998). In contrast, late onset is more common for alcohol dependence among older adults with drinking problems (Zarit & Zarit, 2011). It appears, though, that most people with psychological disorders in late life are experiencing a continuation of symptoms that began earlier.

**FIGURE 11.2** One-year prevalence of psychological disorders by age group in Australia



*Source:* Australian Bureau of Statistics (2007).

Why are rates of psychopathology so low in late life? There are several completely different answers to this question. Above, we described some of the ways that ageing relates to more positive emotionality and more close-knit social circles. Changes like these might enhance mental health as people age. In contrast, some have argued that methodological issues might be leading us to underestimate the prevalence of psychological disorders in late life. We turn to some of these methodological issues next.

## Methodological issues in estimating the prevalence of psychopathology

Methodologically, older adults may be more uncomfortable acknowledging and discussing mental health or drug use problems compared to younger people. In one study, researchers interviewed elderly people about depressive symptoms and then interviewed a family member about whether that elderly person was experiencing depressive symptoms. Among those elderly whom family members described as meeting the criteria for major depressive disorder, about one-quarter did not disclose depressive symptoms to the interviewer (Davison, McCabe, & Mellor, 2009). Discomfort discussing symptoms may minimise prevalence estimates.

In addition to reporting bias, there may be cohort effects. For example, many people who reached adulthood during the drug-oriented era of the 1960s continue to use drugs as they age (Zarit & Zarit, 2011). Their generation is more likely to have problems with substance abuse in late life than previous generations had. For example, although the 50-and-older age group accounted for only 6.6 percent of substance abuse admissions in the United States in 1992, by 2008 the same age group accounted for 12.2 percent of such admissions.

Beyond these explanations, people with psychological disorders are at risk for dying earlier — before age 65 — for several different reasons. Among heavy drinkers, the peak years for death from cirrhosis are between 55 and 64 years of age and cardiovascular disease is also common (Balsis et al., 2007; Shaper, 1990). Cardiovascular disease is also more common among people with a history of anxiety disorders, depressive disorders and bipolar disorder (Kubzansky, 2007). Even milder psychological

disorders compromise immune function and as people age, they become particularly sensitive to these immune effects (Kiecolt-Glaser & Glaser, 2001). This may lead to worse outcomes for many medical conditions that are more common as people age. Psychological disorders are associated with increased mortality (Angst, Stassen, Clayton, & Angst, 2002). For example, Frojd and colleagues (2003) conducted surveys with over 1200 elderly people living in Sweden. Compared to those with low scores, those who obtained high scores on a self-report measure of depression were 2.5 times as likely to die within the next 6 years. Because people with psychological disorders may die earlier, studies on ageing may suffer from the issue of selective mortality.

These three methodological issues — response biases, cohort effects and selective mortality — could help explain the low rates of psychological disorders in late life. Most researchers, however, believe that ageing is also genuinely related to better mental health. Above, we noted that emotional coping improves as people age. This should translate into a decrease in psychological disorders. Some longitudinal studies suggest that many people who experience psychological disorders early in life seem to grow out of those symptoms. For example, longitudinal studies indicate that heavy drinkers tend to drink less as they enter late life (Fillmore, 1987). Findings like these suggest that enhanced coping abilities developed across the life course may help protect people from psychological disorders during late life.

## 11.3 Neurocognitive disorders in late life

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**LEARNING OUTCOME 11.3** Explain the symptoms, aetiology and treatment of cognitive disorders in the elderly.

Most elderly people do not have cognitive disorders. Indeed, one US study found that the prevalence of cognitive impairment has declined among people over the age of 70 in the last 15 years, perhaps because of improvements in diet, medical care and education levels over time (Langa et al., 2008). Nonetheless, cognitive disorders account for more medical costs than any other geriatric condition (Zarit & Zarit, 2011). We will examine two principal types of cognitive disorders: dementia, a deterioration of cognitive abilities, and delirium, a state of mental confusion. For each, we will consider the clinical description as well as causal factors and treatment.

### Dementia

**Dementia** is not a single specific disease. It is an ‘umbrella’ term used to describe a clinical syndrome characterised by progressive deterioration of cognitive abilities to the point that functioning becomes impaired. There are many different types of dementia, including Alzheimer’s disease, frontotemporal dementia, vascular dementia and dementia with Lewy bodies, which we will discuss in this chapter. The nature and progression of symptoms varies based on the type of dementia; but generally the symptoms have a gradual onset, and are progressive and irreversible.

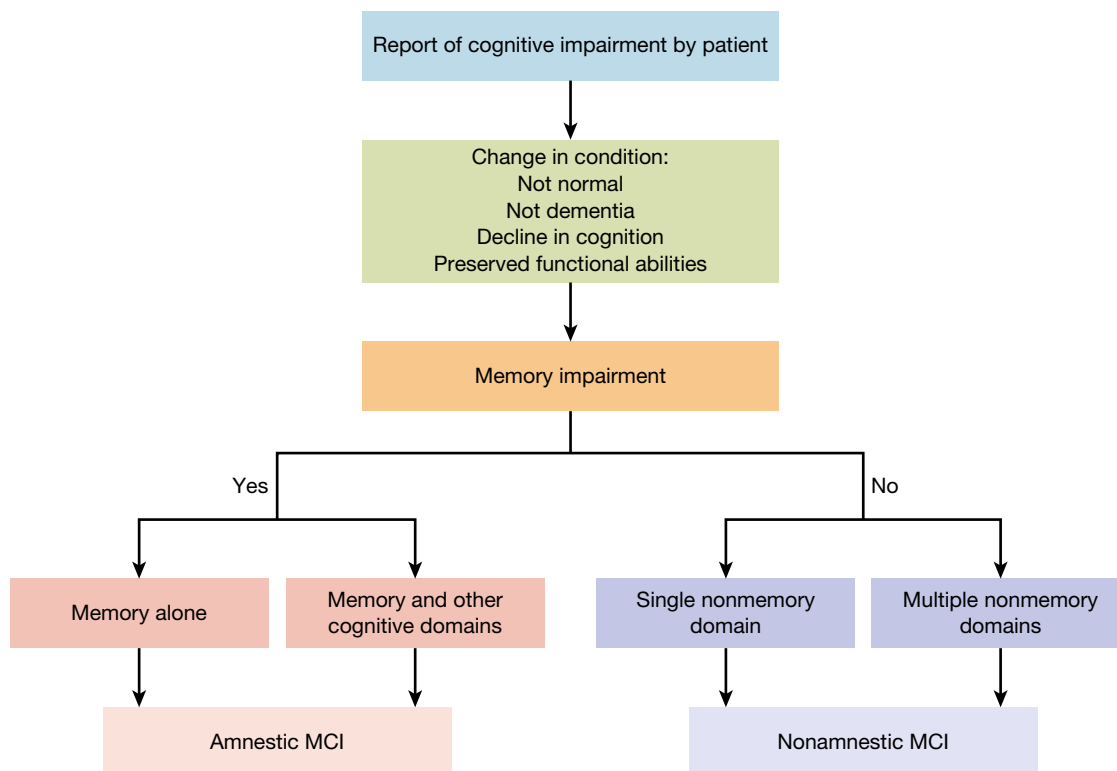
Worldwide prevalence of dementia in 2010 was estimated to be 35.6 million, with numbers expected to double to 65.7 million in 2030 (Prince et al., 2013). The prevalence of dementia increases with advancing age. In Australia, the prevalence of dementia in people aged 65 to 74 is 3.2 percent and increases progressively to 29.5 percent in those aged over 85 years (Australian Institute of Health and Welfare, AIHW, 2012). In 2012, dementia was the third leading cause of death in Australia, following ischaemic heart disease and cerebrovascular disease (ABS, 2012).

Most dementias develop very slowly over a period of years; subtle cognitive and behavioural deficits can be detected well before the person shows any noticeable impairment in function (Small, Fratiglioni, Viitanen, Winblad, & Backman, 2000). For most dementias there is a substantial gap between when symptoms were first noticed and medical help is sought, and a further gap before a condition is actually

diagnosed. One Australian study reported an average time of 1.9 years between when symptoms were first noticed and the first professional consultation, and 3.1 years before a firm diagnosis was made (Speechly, Bridges-Webb, & Passmore, 2008).

**Mild cognitive impairment (MCI)** is a separate diagnostic category used to describe signs of cognitive decline before functional impairment is present. It is sometimes described as a transitional state between the cognition of normal ageing and dementia and is separated into amnesic and non-amnesic mild cognitive impairment (see figure 11.3) (Petersen 2011; Subramanyam & Singh, 2016). Prevalence of mild cognitive impairment ranges from 16 to 20 percent in the general population (Roberts & Knopman, 2013).

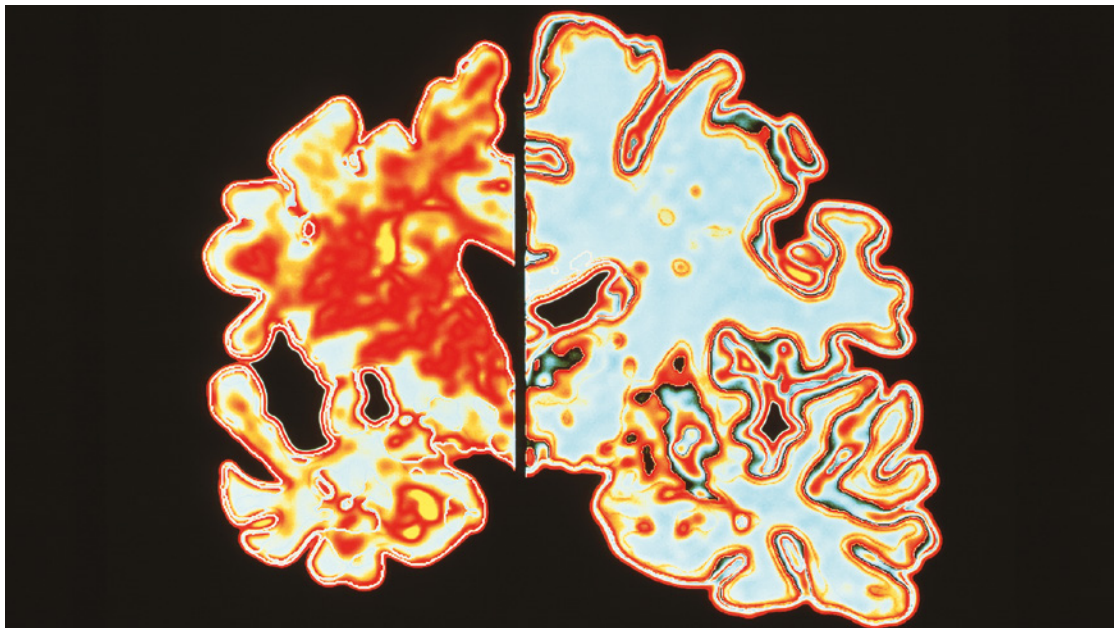
**FIGURE 11.3** Flow chart to guide classification of mild cognitive impairment in a diagnostic setting



Source: Petersen (2011).

Diagnostic criteria for dementia and mild cognitive impairment have been developed by a consensus panel of leading experts with support from the National Institute of Aging and the Alzheimer's Association (Albert et al., 2011; McKhann et al., 2011). The DSM-5 system also provides parallel diagnoses like those for dementia and for mild cognitive impairment. See table 11.3 for an overview of the DSM-5 diagnoses. DSM mild neurocognitive disorders are similar to mild cognitive impairment, whereas DSM major neurocognitive disorders are similar to a diagnosis of dementia. Throughout this chapter, we use the terms *dementia* (rather than *major neurocognitive disorder*) and *mild cognitive impairment* (rather than *mild neurocognitive disorder*).

Computer-generated images of a brain of a person with Alzheimer's disease and a healthy brain. Note that the diseased brain (left) has shrunk considerably owing to the loss of nerve cells.



**TABLE 11.3** DSM-5 neurocognitive disorders

**Neurocognitive disorder: Specify mild or major**

Neurocognitive disorder associated with:

- Alzheimer's disease
- frontotemporal lobar degeneration
- vascular disease
- traumatic brain injury
- Lewy body disease
- Parkinson's disease
- HIV infection
- substance/medication use
- Huntington's disease
- prion disease.

Neurocognitive disorder due to:

- another medical condition
- multiple aetiologies.

**DSM-5**

**DSM-5 criteria for mild neurocognitive disorder**

- People with mild neurocognitive disorder experience modest cognitive decline from previous levels in one or more domains (e.g., complex attention, memory, language, executive function, social cognition) based on both of the following:
  - concerns of the patient, a close other or a clinician
  - modest neurocognitive decline on formal testing or equivalent clinical evaluation.
- The cognitive deficits do not interfere with independence in everyday activities (e.g., paying bills or managing medications), even though greater effort, compensatory strategies or accommodation may be required to maintain independence.
- The cognitive deficits do not occur exclusively in the context of delirium and are not due to another psychological disorder.

## DSM-5

### DSM-5 criteria for major neurocognitive disorder

- People with major neurocognitive disorder experience significant cognitive decline from previous levels in one or more domains (e.g., complex attention, memory, language, executive function, social cognition) based on both of the following:
  - concerns of the patient, a close other or a clinician
  - substantial neurocognitive impairment, preferably documented by standardised neuropsychological testing or equivalent clinical evaluation.
- The cognitive deficits interfere with independence in everyday activities.
- The cognitive deficits do not occur exclusively in the context of delirium and are not due to another psychological disorder.

There is some debate about where to draw the line between mild cognitive impairment and dementia, as well as how early to diagnose mild cognitive impairment. The DSM-5 distinguishes between mild and major neurocognitive disorder based on whether symptoms interfere with the ability to live independently. The DSM-5 criteria for mild neurocognitive disorder require a low score on only one cognitive test. This may lead to an artificially high rate of diagnosis for mild neurocognitive disorder; requiring low scores on at least two different cognitive tests could drastically decrease the rates of diagnosis (Jak et al., 2009).

It is important to note that not all individuals diagnosed with mild cognitive impairment develop dementia. Among adults with mild cognitive impairment, about 10 percent per year will develop dementia; in comparison to 1 percent per year for individuals without mild cognitive impairment (Bischkopf, Busse, & Angermeyer, 2002). It is important to provide careful psychoeducation regarding these diagnoses so that patients and family members do not assume that symptoms will necessarily progress.

In the next section, we will discuss four different types of dementia; Alzheimer's disease, frontotemporal dementia, vascular dementia and dementia with Lewy bodies. Then we will turn to discussing the causes and treatment of dementia.

### Alzheimer's disease

**Alzheimer's disease** was initially described by the German neurologist Alois Alzheimer in 1906. It is the most common type of dementia accounting for approximately 60 to 80 percent of cases (Terry, 2006). Death usually occurs within 12 years after the onset of symptoms, with mean survival rates of 8.7 years for those diagnosed at the age of 65 years and 3.4 years for those diagnosed at the age of 90 years (Brookmeyer, Corrada, Curriero, & Kawas, 2002).

The most common symptom of Alzheimer's disease is memory loss. In the early stage of the disease, memory impairment for recent events is common whereas long-term memory remains intact. The illness may begin with absentmindedness and gaps in memory for new material. The individual may have trouble recalling recent events and conversations, have trouble coming up with the right word or name and forgetting information one has just read. The person may leave tasks unfinished and forgotten if interrupted and misplace objects. These shortcomings may be overlooked for several years but eventually interfere with daily living.

## CLINICAL CASE

### Ellen

'I am so glad you came,' Ellen says when I greet her. She is sitting at the dining room table sipping juice, a slender, almost frail woman. But Ellen has presence. She has the posture of a dancer: shoulders back, neck elongated, head up, and the gaunt face of a once beautiful woman, with large milky hazel eyes and high patrician cheekbones. She smiles and reaches for my hand. 'It is so nice of you to visit,' she says.

Ellen is gracious and polite, but the truth is, she doesn't remember me. She doesn't remember that we've visited a half dozen times before, that a few days ago we had tea together, that just yesterday I sat on her bed for a half hour massaging her hands with rosemary mint lotion. Ellen, like the 43 others living at this residential care facility, has Alzheimer's disease. Her short-term memory is shot, and her long-term memory is quirky and dreamlike, with images that are sometimes bright and lucid, and other times so out of focus that she can hardly make them out. Her life is like a puzzle someone took apart when she wasn't looking. She can see some of the pieces, but she can no longer see how they fit together. (Quoted from Kessler, August 22, 2004, p. 1)

### QUESTIONS

1. What is the most common symptom of Alzheimer's disease?
2. Why might Ellen have difficulties living at home?

Memory loss is not the only symptom of Alzheimer's disease. Apathy is common even before the cognitive symptoms become noticeable (Balsis, Carpenter, & Storandt, 2005) and about a third of people develop full-blown depression as the illness worsens (Vinkers, Gussekloo, Stek, Westendorp, & van der Mast, 2004). As the disease develops, problems with language skills and word finding intensify. Visual-spatial abilities decline, which can be manifested in **disorientation** (confusion with respect to time, place or identity). The person may easily become lost, even in familiar surroundings. The person may also have difficulties with reasoning, problem solving or poor judgement.

As the brain deterioration progresses, the range and severity of behavioural symptoms increase. People with the disorder may be unaware of their cognitive problems and they may blame others for lost objects even to the point of developing delusions of being persecuted. Memory continues to deteriorate and the person becomes increasingly disoriented and agitated. As the dementia progresses, a parent is unable to remember the name of a daughter or son and later may not even recall that he or she has children or recognise them when they come to visit. The person may forget to bathe or dress adequately. Sleep may be disrupted and the person may sleep during the day and be restless at night. Judgement may become faulty and the person may have difficulty comprehending situations and making plans or decisions. In the terminal phase of the illness, personality loses its sparkle and integrity. Relatives and friends say that the person is just not himself or herself anymore. Social involvement with others keeps narrowing. Finally, the person is oblivious to his or her surroundings.

### CLINICAL CASE

#### Sophie

Sophie was 51 when she was diagnosed with Alzheimer's disease. At her funeral five years later, her daughter described her as 'a strong, vital woman, a fine journalist and radio host who kept everyone on their toes, and an irreplaceable mum'. Sophie was not the first person in her family to be diagnosed with Alzheimer's disease. Sophie's mother was diagnosed with the disease and was cared for by Sophie for several years before she died. It was a difficult time for the family, as they struggled with the tragedy of watching Sophie's mother become disinhibited, verbally abusive and eventually mute and incontinent. It was then that Sophie researched the disorder to raise public awareness of the impact Alzheimer's disease has on families.

So, when Sophie noticed changes in her memory at the age of 49, she became concerned. She found herself forgetting the beginning of books before she finished reading them, she had to re-read articles she had written only a couple of days ago to remind herself of the content and she would frequently forget people's names or important dates. As a journalist, she prided herself on her good memory and ability to complete entire interviews without taking notes. Friends and family told her that this level of forgetfulness was normal as we age and recounted a number of their own memory lapses.

In the next 18 months, it became apparent to Sophie and her family that something was wrong. Sophie had to retire as a radio talk show host because she struggled to find the right words in conversation. In interviews, she would also sometimes repeat questions she asked a few minutes before and would forget the name of the person she was talking to. (Adapted from Ogden, 2005)

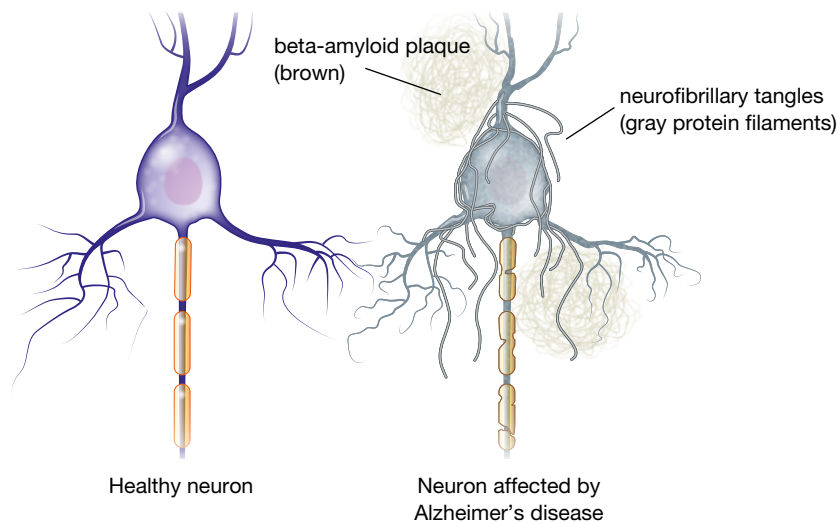
### QUESTIONS

1. What might have Sophie initially attributed her symptoms to?
2. In the case study above, which information helps you distinguish between dementia versus mild cognitive impairment?

People with Alzheimer's disease have more **plaques** (small, round beta-amyloid protein deposits that are outside the neurons) and **neurofibrillary tangles** (twisted protein filaments composed largely of the protein tau in the axons of neurons) than would be expected for the person's age (see figure 11.4). Some people produce excessive amounts of beta-amyloid, whereas others seem to have deficiencies in the mechanisms for clearing beta-amyloid from the brain (Jack et al., 2011). The beta-amyloid plaques are most densely present in the frontal cortex (Klunk et al., 2004) and they may be present for 10 to 20 years before the cognitive symptoms become noticeable. Plaques can be measured using a specialised type of PET scan. Tangles are most often measured in cerebrospinal fluid, although they can be measured using a PET scan as well. Tangles are most densely present in the hippocampus, an area that is important for memory. Over time, as the disease progresses, plaques and tangles spread through more of the brain (Klunk et al., 2004; Sperling et al., 2011).

**FIGURE 11.4**

People with Alzheimer's disease have more plaques (small, round beta-amyloid protein deposits that are outside the neurons) and neurofibrillary tangles (twisted protein filaments composed largely of the protein tau in the axons of neurons) than would be expected for the person's age.

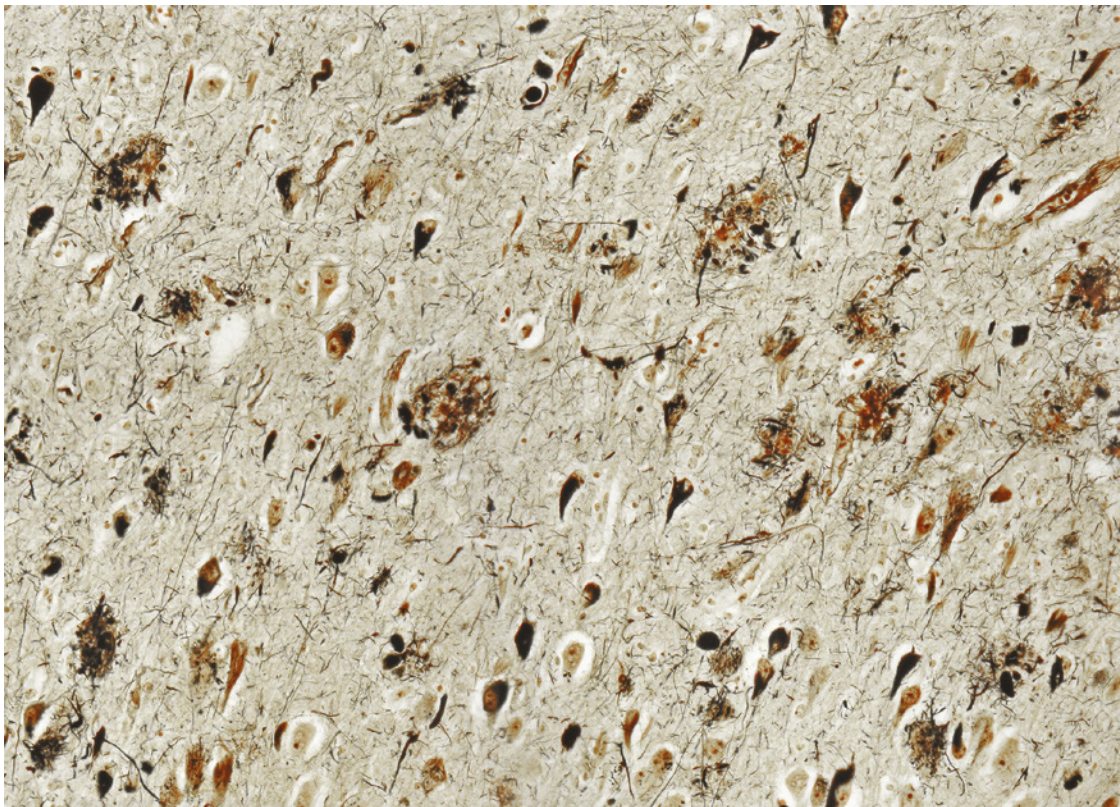


Immune responses to plaques lead to inflammation (Gorelick, 2010), which then triggers a series of brain changes over time, as shown in figure 11.5. At early stages, there seems to be a loss of synapses for acetylcholinergic (ACh) and glutamatergic neurons (Selkoe, 2002). Neurons also begin to die. As

neurons die, the entorhinal cortex and then the hippocampus and other regions of the cerebral cortex shrink, and later the frontal, temporal and parietal lobes shrink. As this happens, the ventricles become enlarged (see figure 11.6). The cerebellum, spinal cord and motor and sensory areas of the cortex are less affected, which is why people with Alzheimer's do not appear to have anything physically wrong with them until late in the disease process. For some time, people with Alzheimer's are able to walk around normally and their overlearned habits, such as making small talk, remain intact, so that in short encounters strangers may not notice anything amiss. About 25 percent of people with Alzheimer's disease eventually develop brain deterioration that leads to motor deficits.

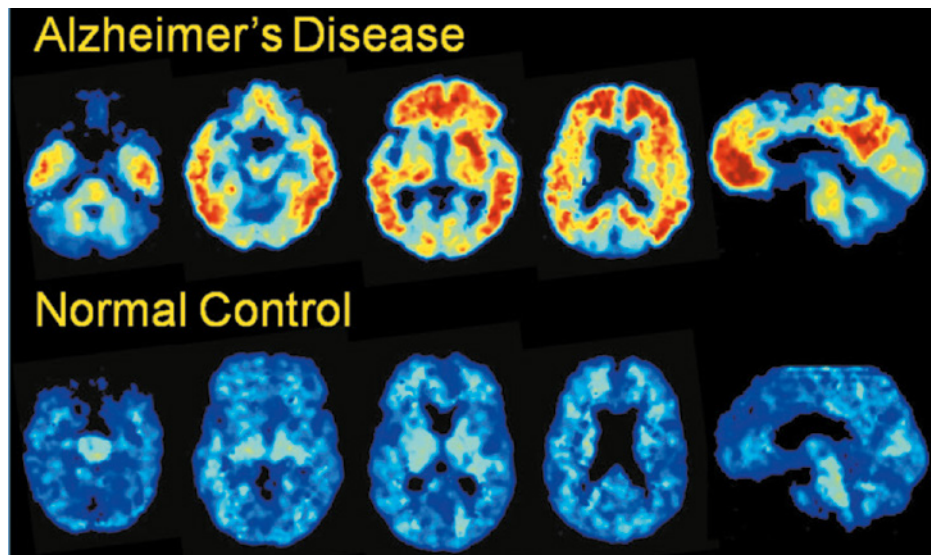
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Light micrograph of a section of entorhinal cortex brain tissue affected by Alzheimer's disease showing amyloid plaques (rounded brown agglomerations) and neurofibrillary tangles (dark teardrop shapes). In Alzheimer's disease, neurons die in the entorhinal cortex and then the loss of neurons begins to occur in other regions of the brain.

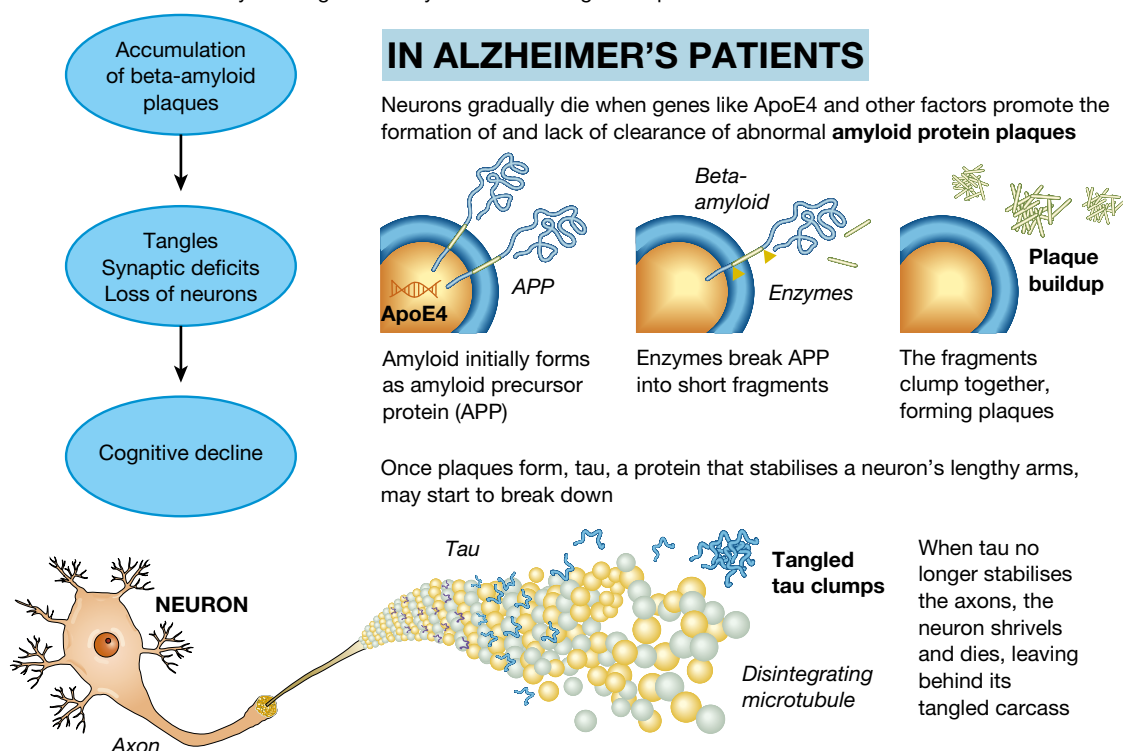


By far, the genetic polymorphism with the largest contribution to Alzheimer's disease is a polymorphism of a gene on chromosome 19, called the apolipoprotein  $\epsilon 4$  or *ApoE-4* allele. Whereas having one  $\epsilon 4$  allele increases the risk of Alzheimer's disease to about 20 percent, having two  $\epsilon 4$  alleles brings the risk substantially higher. Researchers are beginning to understand some of the ways that the  $\epsilon 4$  may increase risk of the disorder.  $\epsilon 4$  appears to interfere with clearing excess beta-amyloid peptide from the brain. People with two of the  $\epsilon 4$  alleles show overproduction of beta-amyloid plaques, loss of neurons in the hippocampus and low glucose metabolism in several regions of the cerebral cortex even before they develop symptoms of Alzheimer's disease (Bookheimer & Burggren, 2009).

Positron emission tomography (PET) images of the brain after administration of Pittsburgh Compound B (Dugger et al., 2002) in a woman with Alzheimer's disease shows high levels of amyloid plaque (upper series). In contrast, low levels of amyloid plaque are seen in the PET images of a woman with no Alzheimer's symptoms (lower series).

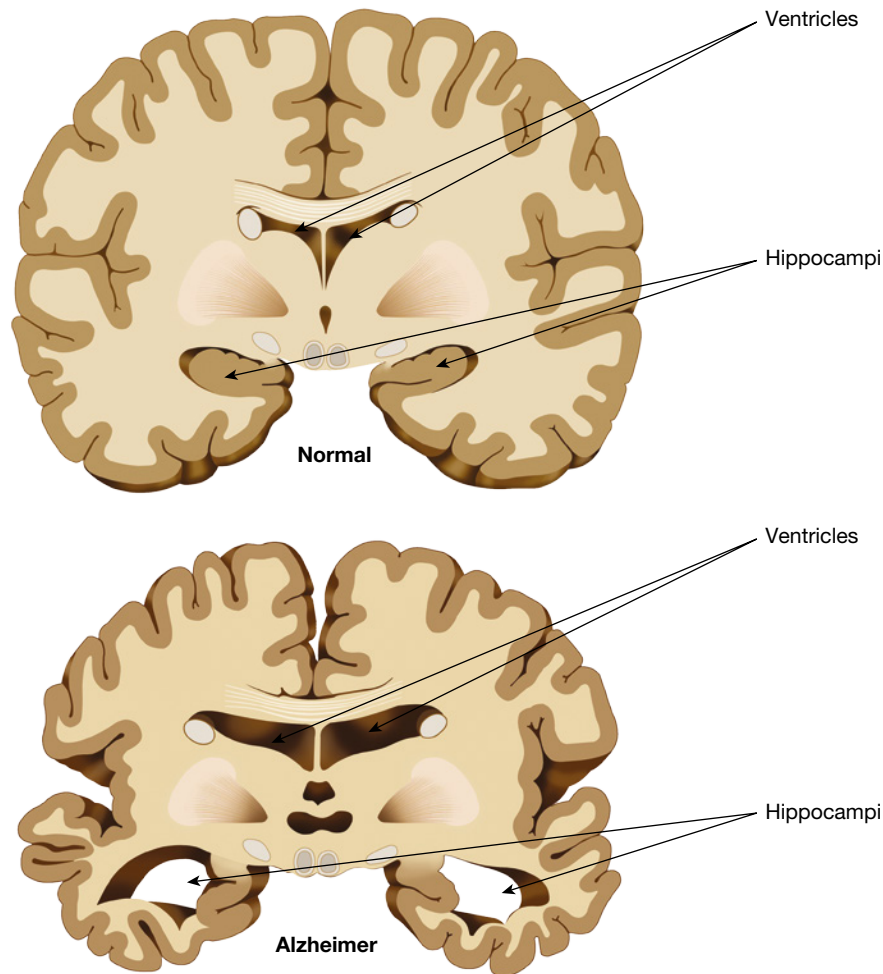


**FIGURE 11.5** In Alzheimer's disease, risk factors such as the gene *ApoE-4* lead to an increased deposition of beta-amyloid plaques. These plaques build up outside of the neurons for 10 to 20 years before the cognitive symptoms are noticeable. Neurofibrillary tangles of tau form and the neurons die. To prevent the death of neurons, researchers hope to develop treatments that will address as many of the genetic amyloid and tau targets as possible.



**FIGURE 11.6**

Schematic illustration of the dissection of a normal brain compare to one with Alzheimer's disease. As demonstrated in the diagram, Alzheimer's disease results in overall cortical shrinkage, enlarged ventricles and atrophy of the hippocampi.



Many of the genes that increase risk for Alzheimer's disease are related to immune function and cholesterol metabolism (Jones et al., 2010). Immune processes and excessively high cholesterol can trigger inflammation and accordingly, conditions that involve immune and inflammatory processes also appear related to a greater risk of Alzheimer's disease. For example, type II diabetes, which has been tied to immune and inflammatory changes, is related to greater risk of developing Alzheimer's disease (Ferreira, Clarke, Bomfim, & De Felice, 2014). Similarly, brain traumas from accidents or injuries can also increase the risk of Alzheimer's disease later in life (Fleminger, Oliver, Lovestone, Rabe-Hesketh, & Giora, 2003).

Beyond genes, lifestyle variables may play a role in Alzheimer's. For example, smoking, being single, obesity, depression and low social support are related to a greater risk of Alzheimer's disease, while a Mediterranean diet, exercise, education and engagement in cognitive activities are related to a lower risk (Lieb et al., 2009; Williams, Plassman, Burke, Holsinger, & Benjamin, 2010).

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Exercise programs are of some help in reducing the risk and severity of Alzheimer's disease.



In one study, lifestyle effects were studied in 2509 elderly people who were enrolled in the study during their 70s and followed for eight years. Those with a high school education who exercised at least once a week, remained socially active and did not smoke sustained their cognitive functioning without decline throughout the eight years (Yaffe et al., 2009). Of the various lifestyle factors, the effects for exercise, cognitive engagement and depression have received a good deal of study and so we focus on those here.

Several studies suggest that exercise may predict fewer memory problems. Regular exercise predicts less decline in cognitive functions (Sofi, Valecchi, Bacci et al., 2011) and as shown in a meta-analysis of 163 797 participants, it also strongly predicts decreased risk of developing Alzheimer's disease over time (Hamer & Chida, 2009). Exercise levels at mid-life seem important, but so does sustaining exercise as one enters late life. Exercise has been related to lower levels of plaques in the brain and particularly so for those with the *ApoE-4* polymorphism (Head et al., 2012).

Engagement in intellectual activities also appears helpful, with some proposing a 'use it or lose it' model of Alzheimer's. For example, regular reading of a newspaper is related to lower risk (Wilson, Scherr, Schneider, Tang, & Bennett, 2007). Findings of a meta-analysis including 29 000 persons drawn from 22 representative community samples suggested that frequent cognitive activity (e.g., reading and puzzle solving) is related to a 46 percent decrease in risk of Alzheimer's disease compared to infrequent cognitive activity (Valenzuela & Sachdev, 2006). Parallel with the findings for exercise, engagement in intellectual activities protects against cognitive decline for those with the *ApoE-4* polymorphism (Vemuri et al., 2014).

Intriguingly, among people with similar levels of plaques and tangles in their brain, those with higher levels of intellectual activity show fewer cognitive symptoms. That is, intellectual activity seems to protect against the expression of underlying neurobiological disease (Wilson, et al., 2007). This type of work has led to the concept of **cognitive reserve** or the idea that some people may be able to compensate for the disease by using alternative brain networks or cognitive strategies such that cognitive symptoms are less pronounced.

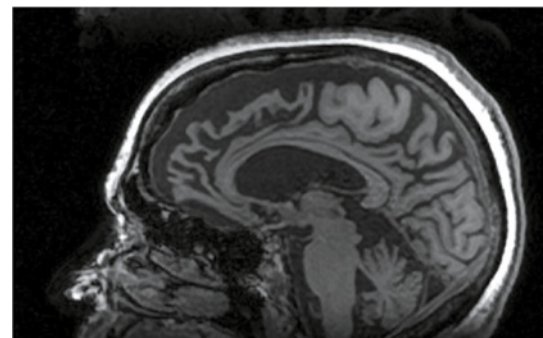
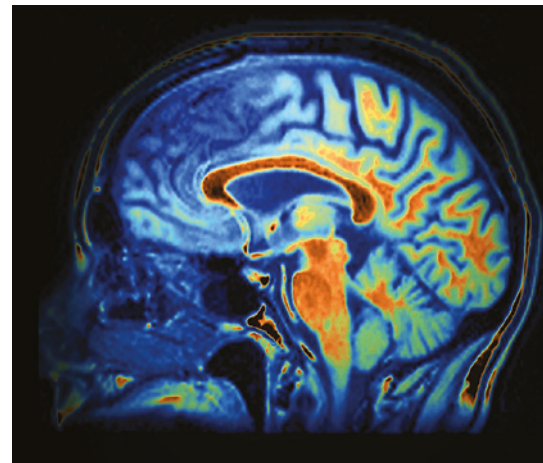
One concern is that naturalistic studies cannot disentangle whether the people who choose to engage in exercise or cognitive activities differ in some important way (on characteristics relevant to disease) from those who do not engage in these activities. We know that biological changes in the brain begin 20 years before the symptoms of Alzheimer's disease first emerge; it is plausible that those brain changes influence motivation to take part in exercise or cognitive activities. The complexity of trying to identify the direction of effects is illustrated by considering the relationship of depression and Alzheimer's disease. We mentioned above that depression can be a consequence of dementia. The opposite direction of effects seems to occur as well: a lifetime history of depression predicts more decline in cognitive functioning (Ganguli, Du, Dodge, Ratcliff, & Chang, 2006), greater risk for Alzheimer's disease and other forms of dementia (Diniz, Butters, Albert, Dew, & Reynolds, 2013) and, among those who develop Alzheimer's disease, a faster progression of the illness (Rapp et al., 2006). These effects seem to be present even when baseline cognitive impairment and other medical issues are controlled (Goveas, Espeland, Woods, Wassertheil-Smoller, & Kotchen, 2011).

### Frontotemporal dementia

As suggested by the name, **frontotemporal dementia (FTD)** is characterised by loss of neurons in frontal and temporal regions of the brain. The neuronal deterioration of FTD occurs predominantly in the anterior temporal lobes and prefrontal cortex (Miller, Ikonte, Ponton, & Levy, 1997). FTD typically begins in the mid- to late 50s and it progresses rapidly; death usually occurs within 5–10 years of the diagnosis (Hu et al., 2009). FTD is rare, affecting less than 1 percent of the population (Pressman & Miller, 2014).

There are multiple subtypes of FTD, separated into a behavioural variant (bvFTD) and language variants. Unlike Alzheimer's disease, memory is not severely impaired in FTD. The most common variant, bvFTD, is characterised by a progressive deterioration of personality, social skills and cognition. Hallmark features of bvFTD include early behavioural disinhibition, such as socially inappropriate behaviour (e.g., kissing strangers, verbal aggression), loss of manners or decorum (e.g., inappropriate laughing, offensive jokes, loss of respect for personal space) and impulsive or careless actions (e.g., reckless driving, new onset gambling). The most common initial symptoms include loss of motivation, reduced interest in previously enjoyed activities and decreased initiation of behaviour. Loss of sympathy or empathy, perseverative behaviour, hyperorality

Frontotemporal dementia. The photo on the top is a coloured magnetic resonance imaging (MRI) scan of the brain of a 50-year-old with frontotemporal dementia. The front of the brain is at the left. The frontal (left) and temporal (centre) lobes have atrophied (shrunk).



(e.g., putting inedible objects in mouth) and dietary changes (i.e., preference for sweet foods) may also occur (Rascovsky et al., 2011; Seelaar, Rohrer, Pijnenburg, Fox, & van Swieten, 2011). In early stages, significant others may notice changes in personality and judgement. For example, the successful and savvy businessman may begin to make terrible investments (Levenson & Miller, 2007). Because a person affected by this disorder might suddenly start to overeat, chain smoke, drink alcohol or demonstrate other behavioural symptoms, FTD is often misdiagnosed as a midlife crisis or as a psychological disorder such as depression, bipolar disorder or schizophrenia (Zhou & Seeley, 2014).

FTD strikes emotional processes more profoundly than Alzheimer's disease does and in doing so, it can damage social relationships. Particular deficits seem to emerge in the ability to regulate emotions (Goodkind, Gyurak, McCarthy, Miller, & Levenson, 2010). This may lead the person to violate social conventions (Mendez, Lauterbach, & Sampson, 2008). Persons with FTD do not seem to notice that they have made a social mistake and so they do not experience embarrassment in contexts where others might. As you might imagine, the changes in personality and emotion, together with a lack of insight, influence relationships (Mendez et al., 2008). Marital satisfaction is more affected by FTD than by Alzheimer's disease (Ascher et al., 2010).

FTD can be caused by many different molecular processes (Mackenzie et al., 2009). One of these is Pick's disease, characterised by the presence of Pick bodies (spherical inclusions) within neurons, but many other diseases or pathological processes can result in FTD. Some people with FTD show high levels of tau, the protein filaments that contribute to the neurofibrillary tangles observed in Alzheimer's disease, but others do not (Josephs, 2008). FTD has a strong genetic component (Cruts et al., 2006).

Some of the language variants of FTD include progressive non-fluent aphasia (PNFA) and semantic dementia. These dementias are characterised by progressive language impairment. In some individuals signs and symptoms may be confined to the domain of language for many years, whereas others experience other cognitive deficits within a few years. Nevertheless, the language dysfunction is the most salient feature and deteriorates most rapidly in the course of the disease (Mesulam, 2003). PNFA is characterised by agrammaticism in language production and effortful speech (Gorno-Tempini et al., 2011). Agrammaticism refers to inappropriate word order, misuse of endings, pronouns and verb tense. Mesulam (2003) provides the following example from an email 'I will come my house in your car and drive my car into Chicago. ... You will get your car and my car park in my driveway, Love Mum' (p. 1537). Semantic dementia is characterised by impaired word comprehension and object naming (Gorno-Tempini et al., 2011; Mesulam, 2013). It is a disorder of conceptual knowledge resulting in a loss of understanding of word meaning.

## CLINICAL CASE

### James

James is a 54-year-old assistant at a residential care facility. He presented to a memory clinic with a 12-month history of memory problems, specifically affecting his ability to recall the names of people and things. His difficulty first became apparent when he realised he could not tell the residents at the facility what foods were available on the meal trolley. He also had great difficulties naming family members, friends and famous personalities. Despite these problems, James' day-to-day and personal autobiographical memory was good. He was able to provide an accurate history of his past life, work and recent family events such as his daughter's birthday party. His family confirmed that there were no changes in personality or behaviour. (Adapted from Hodges, Patterson, Oxbury, & Funnell, 1992)

### QUESTIONS

1. What diagnosis should the clinician consider?
2. Why might these difficulties be interpreted as memory problems?

## CLINICAL CASE

### Paul

Paul is a 63-year-old retired man. He was admitted to hospital for a psychiatric review at the request of his wife. Paul had no complaints; however, his wife reported that his behaviour had changed a few years ago and was becoming progressively worse. About two years ago, he made sexually inappropriate advances towards a work colleague and later a family friend, which caused major commotion in the family. One year ago, he started making speeches and jokes with sexual content, laughing inappropriately and not responding to social cues to stop. He recently started taking long walks and yelling in the street. In contrast, he had introverted periods where he stayed at home. Paul's appetite had increased. Whereas he did not have much of a sweet tooth before, he now started craving sweets and gained 10 kilograms. About a year ago he became forgetful (e.g., locking his keys in the car). On some occasions he became angry and violent for no apparent reason.

In order to eliminate possible reversible dementia causes, the doctors undertook a complete blood test and everything came up clear. Brain magnetic resonance imaging (MRI) showed severe bilateral atrophy of the frontotemporal lobes. (Adapted from Ozten, Hizli, Salcini, Kagan, & Tanridag, 2013)

### QUESTIONS

1. What are the differential diagnoses?
2. What symptoms might be confused with a psychiatric disorder (e.g., bipolar disorder, schizophrenia, depression)?

## Vascular dementia

By definition, **vascular dementia** is caused by cerebrovascular disease. Most commonly, strokes cause a blood clot, which then impairs circulation and results in the death of neurons. About 7 percent of people will develop dementia in the year after a first stroke and the risk of dementia increases with recurrent strokes (Pendlebury & Rothwell, 2009). Risk for vascular dementia involves the same risk factors as those for cardiovascular disease in general — for example, high cholesterol, cigarette smoking and elevated blood pressure (Moroney et al., 1999). Because strokes and cardiovascular disease can strike different regions of the brain, the symptoms of vascular dementia vary a good deal. The onset of symptoms is usually more rapid in vascular dementia than in other forms of dementia. Vascular dementia is the second largest cause of dementia accounting for 15 to 20 percent of cases and co-occurs with Alzheimer's disorder in 50 percent of cases (Lopez et al., 2005; Moorhouse & Rockwood, 2008).

## Dementia with Lewy bodies

In **dementia with Lewy bodies (DLB)**, protein deposits called Lewy bodies form in the brain and cause the cognitive decline. DLB is characterised by fluctuating cognition and alertness, visual hallucinations and spontaneous motor features of parkinsonism. In the early stages of the disease, individuals may fluctuate between global cognitive impairment and near-normal performance. Recurrent visual hallucinations are usually well formed and detailed and often overlap with other perceptual disorders. For example, the person may describe faces emerging from trees, cushions or flowers. DLB is diagnosed when the cognitive symptoms occur before or concurrently with parkinsonian symptoms. In contrast, Parkinson's disease dementia is diagnosed when dementia occurs within the context of well-established Parkinson's disease (Geser, Wenning, Poewe, & McKeith, 2005; McKeith et al., 2005; McKeith et al., 1996).

People with DLB are often extremely sensitive to the physical side effects of antipsychotic medications. Another distinct symptom of DLB is that people often experience intense dreams accompanied by levels of movement and vocalising that may make them seem as though they are 'acting out their dreams' (McKeith et al., 2005).

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Dementia with Lewy bodies is defined by the presence of abnormal deposits called Lewy bodies. The Lewy bodies are found throughout the brain.



### **Dementias caused by disease and injury**

A number of other medical conditions can produce dementia. Encephalitis, a term for any inflammation of brain tissue, is caused by viruses that enter the brain. Meningitis, an inflammation of the membranes covering the outer brain, is usually caused by a bacterial infection. Both encephalitis and meningitis can cause dementia. The organism that produces the venereal disease syphilis can invade the brain and cause dementia. HIV, head traumas, brain tumours, nutritional deficiencies (especially of B-complex vitamins), kidney or liver failure and endocrine problems such as hyperthyroidism can result in dementia. Exposure to toxins (such as lead or mercury) and chronic substance use are both additional causes.

### **Treatments for dementia**

Sadly, despite intensive research in this area, there is no cure for dementia (Williams et al., 2010), although some medications are used to treat it and related syndromes. There are also some psychological and lifestyle approaches to treatment.

#### **Medications**

Because no medications have been shown to help address the cognitive symptoms of FTD (Caselli & Yaari, 2008), we focus on interventions for dementias other than FTD. Much of the treatment research has focused on Alzheimer's disease and on memory decline. Medications help slow decline, but they do not restore memory function to previous levels. The most commonly used medications for dementia are the cholinesterase inhibitors (drugs that interfere with the breakdown of acetylcholine), including donepezil (Aricept) and rivastigmine (Exelon). Cholinesterase inhibitors have a slight effect in slowing

memory decline compared to placebo for those with Alzheimer's (Birks, 2006; Howard et al., 2012) and dementia with Lewy bodies (Maidment, Fox, & Boustani, 2006; McKeith, 2006). In addition to cholinesterase inhibitors, memantine (Namenda), a drug that affects glutamate receptors involved in memory, has shown small effects in placebo-controlled trials for Alzheimer's disease. Unfortunately, many people discontinue these drugs due to aversive side effects such as nausea (Maidment et al., 2006).

Medical treatments are commonly used to address psychological symptoms, such as depression and agitation that commonly co-occur with dementia. For example, antidepressants can help relieve comorbid depressive symptoms in Alzheimer's disease (Modrego, 2010) and FTD (Mendez & Shapira, 2008). Because depression produces more cognitive impairment in the elderly than it does in younger people (Lockwood, Alexopoulos, Kakuma, & van Gorp, 2000), treating depressive symptoms can often lead to improvements in cognitive symptoms. Although antipsychotic medications can provide modest relief for aggressive agitation (Lonergan, Britton, & Luxenberg, 2007), they also increase the risk of death among elderly people with dementia (Food and Drug Administration, 2005).

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Hazel Hawke, the first wife of Australian Prime Minister Bob Hawke, publicly announced in 2003 that she had been diagnosed with Alzheimer's disease. She made many public appearances hoping to raise awareness of the disease and raise money for the Hazel Hawke Alzheimer's Research and Care Fund. Hazel stated the following about the disease: 'I feel as if . . . my autonomy has been taken. I have an illness. I've acknowledged it. I manage it as best I can. I've been told in no uncertain terms . . . that it will progress. If it does, I think I would be aware enough to know that it had progressed' (Grattan, 2003). Hazel died in 2013 at the age of 83 years.



Some of the disappointments in developing treatments have led to new ways of thinking about these disorders. For example, for some time, researchers were striving to find ways to remove plaques from the brains of people with Alzheimer's disease. Surprisingly, when researchers developed a medication that removed beta-amyloid plaques, they found that cognitive deficits continued and even worsened after the plaques were removed (Holmes et al., 2008). Remember, though, that beta-amyloid plaques accumulate in the brain for years before symptoms are observable among people with Alzheimer's disease. By the time people had been diagnosed and had begun to receive the intervention, biological disease processes had already been occurring for years. Findings like these have led researchers to focus more on prevention. One way to do this is to study factors that reduce the chances of mild cognitive impairment becoming a dementia. Another way is to study people who have early biological markers indicating risk for Alzheimer's disease, such as signs of

plaques, tangles and neuronal death (Sperling et al., 2011). Researchers are testing a host of interventions to target the development of plaques and tangles, as well as the associated immune processes (Gandy, 2014). Current thinking is that the best hope for future treatments may be treating the biological processes before symptoms emerge (Sutphen, Fagan, & Holtzman, 2014).

### Psychological and lifestyle treatments

Therapy can help families and patients cope with the diagnosis, discuss symptoms of dementia and provide support. Generally, the therapist allows opportunities for the person with dementia and the family to discuss the diagnosis. The therapist also provides accurate information about the diagnosis and helps family members care for the person in the home (Knight, 1996). See focus on discovery 11.1 for more detail on treatments offered to support caregivers.

Interventions to increase exercise also have modest benefits in improving cognitive function. In a meta-analysis of studies including 824 people, exercise programs were shown to improve cognitive functioning for those with mild to moderate cognitive deficits (Heyn, Abreu, & Ottenbacher, 2004). Exercise programs have also been shown to improve cognitive functioning among those already diagnosed with Alzheimer's disease (Forbes, Thiessen, Blake, Forbes, & Forbes, 2013).

Cognitive training programs that focus on improving memory, reasoning or cognitive processing speed have shown modest benefits for older adults. These benefits seem to be specific to the area involved in the training — for example, memory training might help enhance memory but does not seem to help reasoning abilities (Rebok et al., 2014). Cognitive training tasks are more efficacious in individuals with mild cognitive impairment, whereas evidence of efficacy in people diagnosed with dementia is weak (Hill et al., 2016). To help skills generalise more, several researchers have begun to focus on how to teach 'meta-cognitive' skills — that is, ways to think about thinking. As an example of the success of these meta-cognitive programs, teaching people strategies for enhancing memory seems to improve performance across a range of tasks (Hertzog, Kramer, Wilson, & Lindenberger, 2009). Similarly, training in multitasking appeared to

Computerised training programs have been developed with the aim of enhancing cognitive skills. Companies producing these training programs report tens of millions of users.



Providing memory aids is one way of compensating for memory loss.



improve not just ability to multitask, but also ability to hold items in memory and to sustain attention to a task (Anguera et al., 2013).

Behavioural approaches have been shown to help compensate for memory loss and to reduce depression and disruptive behaviour among people with early stages of dementia. For example, external memory aids such as shopping lists, calendars, phone lists and labels can help when placed prominently as visual reminders (Buchanan, Christenson, Houlihan, & Ostrom, 2011). Pleasant and engaging activities can be increased as a way of diminishing depression (Logsdon, McCurry, & Teri, 2007). Triggers for disruptive behaviour can be identified and changed. Music may help reduce agitation and disruptive behaviour while it is being played (Livingston, Johnston, Katona, Paton, & Lyketsos, 2005). For a review of non-pharmacological interventions, see Oliveira et al., 2015. These behavioural interventions can provide important alternatives to pharmacological approaches.

### FOCUS ON DISCOVERY 11.1

#### Support for caregivers

For every person with dementia who is living in permanent residential aged care, there are at least two living in the community, usually supported by a family (typically by spouses or children). Caregiving for dementia requires much more time than caregiving for most other disorders (Ory, Hoffman, Yee, Tennstedt, & Schulz, 1999) and has been shown to be extremely stressful across a number of cultures (Torti, Gwyther, Reed, Friedman, & Schulman, 2004). Caregivers are at heightened risk for clinical depression and anxiety (Dura, Stukenberg, & Kiecolt-Glaser, 1991), physical illness (Vitaliano, Zhang, & Scanlan, 2003) and decreased immune functioning (Kiecolt-Glaser, Dura, Speicher, & Trask, 1991) compared to non-caregivers.

Families can be helped to cope better with the daily stress of caregiving. For example, because people with Alzheimer's have difficulty placing new information into memory, they can engage in a reasonable conversation but forget the discussion within a few minutes. A caregiver may become impatient unless he or she understands that this impairment is an expected consequence of the brain damage. Family members can learn communication strategies to adapt to the memory loss. For example, families can ask questions that embed the answer. It is much easier to respond to 'Was the person you just spoke to on the phone Harry or Tom?' than to 'Who just called?'

It is also useful for caregivers to understand that patients do not always recognise their limitations and may try to engage in activities beyond their abilities. Caregivers must set limits regarding dangerous activities. For example, caregivers often need to tell their relative that they are no longer able to drive and implement strategies to reduce the behaviour (e.g., hiding the keys).

Caring for a relative with Alzheimer's disease can be stressful. There are several organisations that provide support to carers in different forms (i.e., counselling, support groups, respite).



Programs that teach coping strategies for the caregivers (e.g., increasing pleasant activities, exercise or social support) as well as individual behavioural therapy programs have been shown to relieve caregiver burden (Selwood, Johnson, Katona, Ilyktsos, & Livingston, 2007) and depression (Mittelman, Brodaty, Wallen, & Burns, 2008). Programs lasting at least six weeks (Selwood et al., 2007) or offering multiple components (e.g., psychoeducation about dementia, case-management services and cognitive behavioural strategies) more consistently reduce caregivers' distress (Acton & Kang, 2001). Because caregivers are so powerfully affected, it is recommended that they be given respites from their task. To give the caregiver a break, the person with dementia is sometimes placed in residential respite care for a period of time; or respite workers will travel to the home for a shorter duration. Caregiver support programs have been found to improve the immune function of caregivers (Garand et al., 2002), decrease the medical costs of the person with dementia and slow the timing of institutionalisation (Teri et al., 2003).

### QUESTIONS

1. Given what you now know about dementia, what types of supports would a caregiver need to provide on a daily basis?
2. What types of community support may decrease caregiver burden?

### RESEARCH EXAMPLE

#### Tailored activities program

Behavioural and psychological symptoms of dementia (BPSD) are often the most challenging aspects of dementia care; they lead to increased caregiver burden, greater health care costs and placement into residential care facilities (O'Connor et al., 2014). The tailored activities program (TAP) has been developed in the United States with promising results (Gitlin et al., 2008) and a randomised trial has commenced in Australia (O'Connor et al., 2014). The TAP includes a detailed individual program based on the person's strengths and interests, and focuses on enjoyable activities (e.g., woodwork, puzzles). Very specific step-by-step instructions are provided to the caregiver to help support the individual in engaging in these activities. The first evaluation of the intervention indicated that at the four-month follow-up, caregivers of individuals with dementia participating in the intervention reported reduced frequency of problem behaviours, greater activity engagement and reduced agitation when compared to controls. The carers themselves reported less time being 'on duty' and greater ability to simplify information to the person's needs (Gitlin et al., 2008). Implementation and evaluation of this intervention in the Australian sample is greatly anticipated. The findings may address a gap in the current community support systems for people living with dementia and their caregivers (O'Connor et al., 2014).

### QUESTION

Undertake your own research into the tailored activities program. Has the trial been successful in Australia?

## Delirium

The term **delirium** is derived from the Latin words *de*, meaning 'out of,' and *lira*, meaning 'track'. The term implies being off-track or deviating from the usual state (Wells & Duncan, 1980). As illustrated in the opening scenario of Joan at the beginning of this chapter, delirium is typically described as a clouded state of consciousness. It is of an acute onset, usually hours to days, and is characterised by fluctuating consciousness, attention and cognition. The diagnosis of delirium is clinically based and depends on the presence or absence of certain features; and occurs in the setting of a medical condition or substance intoxication or withdrawal (APA, 2013; Fong, Tulebaev, & Inouye, 2009).

There are many clinical presentations of delirium, which can be broadly classified into hyperactive, hypoactive and mixed. Individuals experiencing a hyperactive delirium may be restless, agitated, aggressive and hypervigilant. Psychotic symptoms such as hallucinations and delusions can also occur. In contrast, patients with hypoactive delirium may be withdrawn, quiet, lethargic, drowsy and respond

slowly to stimuli. This type of delirium is often missed or misdiagnosed as a depression or dementia. When an individual demonstrates features of both hyper and hypoactive delirium, it is termed a mixed delirium (Department of Human Services, DOHS, 2006; Fong et al., 2009).

Typical features include trouble focusing attention and profound disturbances in the sleep/wake cycle (Meagher, 2007). Patients, sometimes rather suddenly, have so much trouble focusing attention that they cannot maintain a coherent stream of thought. They may have trouble answering questions and may not be able to engage in conversation because their mind wanders. As the sleep/wake cycle becomes disturbed, patients become drowsy during the day, yet awake and agitated at night. Vivid dreams and nightmares are common. Some people with delirium may become so disoriented that they are unclear about what day it is, where they are and even who they are. Memory impairment, especially for recent events, is common.

Perceptual disturbances are frequent in delirium. People mistake the unfamiliar for the familiar; for example, they may state that they are at home instead of in a hospital. Although visual hallucinations are common, they are not always present. Delusions — beliefs contrary to reality — have been noted in about 25 percent of older adults with delirium (Camus et al., 2000). These delusions tend to be poorly worked out, fleeting and changeable.

#### DSM-5

##### DSM-5 criteria for delirium

- People with delirium experience a disturbance in attention and awareness.
- People with delirium experience a change in cognition, such as disturbance in orientation, language, memory, perception or visuospatial ability, not better accounted for by a dementia.
- Individuals experience rapid onset (usually within hours or days) and fluctuation during the course of a day.
- Symptoms are caused by a medical condition, substance intoxication or withdrawal, or toxin.

Swings in activity and mood accompany these disordered thoughts and perceptions. People with delirium may shift rapidly from one emotion to another, fluctuating between depression, anxiety, fright, anger, euphoria and irritability. Fever, flushed face, dilated pupils, tremors, rapid heartbeat, elevated blood pressure and incontinence of urine and faeces are common. If delirium worsens, the person may become lethargic and even unresponsive (Webster & Holroyd, 2000). In the course of a 24-hour period, people with delirium have lucid intervals in which they become alert and coherent. Symptoms are usually worse during sleepless nights. These daily fluctuations help distinguish delirium from other syndromes, especially Alzheimer's disease.

People of any age are subject to delirium, but it is more common among children and older adults. Among older adults, it is particularly common in nursing homes and hospitals. One study found that 6 to 12 percent of nursing home residents developed delirium in the course of 1 year (Katz, Parmelee, & Brubaker, 1991) and delirium affects an estimated 14 to 56 percent of hospitalised older adults (Fong et al., 2009). Incidence is even higher when we consider specialised populations; for example, up to 20 percent in general medical admissions experience delirium in contrast to 55 percent in surgical inpatients and 87 percent in intensive care units (Fong et al., 2009; DOHS, 2006).

One risk factor for delirium is a previous diagnosis of dementia, with two-thirds of all cases of delirium in older adults occurring in people diagnosed with dementia. Other risk factors include older age, history of delirium, immobility, multiple comorbidities, chronic disease, sensory impairment, depression, certain medications and sustained sleep deprivation (Ahmed, Leurent, & Sampson, 2014; Fong et al., 2009).

Unfortunately, delirium is often misdiagnosed (Knight, 1996). For example, among 77 hospitalised older adults with clear symptoms of delirium, about 60 percent had no notation of delirium in their hospital chart (Lauril, Pitkala, Strandberg, & Tilvis, 2004). Physicians are particularly unlikely to detect delirium when lethargy is present (Cole, 2004). Delirium is also often misdiagnosed when a person has dementia. Table 11.4 compares the features of dementia and delirium. Consider this suggestion for distinguishing delirium from dementia:

The clinical ‘feel’ of talking with a person with delirium is rather like talking to someone who is acutely intoxicated or in an acute psychotic episode. Whereas the demented patient may not remember the name of the place where she or he is, the delirious patient may believe it is a different sort of place altogether, perhaps mistaking a psychiatric ward for a used car lot. (Knight, 1996, pp. 96–97)

Detecting and treating delirium is of fundamental importance. Untreated, the mortality rate for delirium is high; more than one-third of people with the condition die within a year (McCusker, Cole, & Abrahamowicz, 2002). Beyond the risk of death, elderly adults who develop delirium in the hospital are at an increased risk of further cognitive decline (Jackson, Gordon, Hart, Hopkins, & Ely, 2004) and of being transferred to permanent care (Witlox et al., 2010). It is not clear why delirium predicts such bad outcomes; some believe that delirium may be an indicator of an underlying frailty that becomes apparent in the face of medical conditions.

**TABLE 11.4** Comparative features of dementia and delirium

Dementia	Delirium
Gradual deterioration of abilities	Rapid onset
Most commonly, deficits in memory for recent events	Trouble concentrating and staying with a train of thought
Caused by disease processes that are directly influencing the brain	Secondary to another medical condition
Usually progressive and non-reversible	Symptoms fluctuate over the course of a day
Treatment offers only minimal benefit	Usually reversible by treating underlying condition but potentially fatal if cause — e.g., infection or malnutrition — is not treated
Prevalence increases with age	Prevalence is highest in the very young as well as the old

## Aetiology of delirium

As noted in the diagnostic criteria, delirium is caused by medical conditions. Several causes of delirium have been identified: drug intoxications and drug-withdrawal reactions, metabolic and nutritional imbalances (as in uncontrolled diabetes, thyroid dysfunction, kidney or liver failure, congestive heart failure or malnutrition), dehydration, infections or fevers (like pneumonia or urinary tract infections), neurological disorders (like dementia, head trauma or seizures) and the stress of major surgery (Zarit & Zarit, 2011). One of the most common triggers of delirium is hip surgery (Marcantonio, Flacker, Wright, & Resnick, 2001).

Why are older adults so vulnerable to delirium? Many explanations have been offered: notably, the physical declines of late life, the increased susceptibility to chronic diseases, the many medications prescribed for older people and the greater sensitivity to drugs.

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Medication misuse, whether deliberate or inadvertent, can be a serious problem among older people and can cause delirium.



### **Treatment of delirium**

Development of a delirium frequently depends on a combination of predisposing (e.g., dementia) and participating risk factors (e.g., medications, infections). Complete recovery from delirium is possible if the underlying cause is treated promptly and effectively. Physicians must consider all possible reversible causes of the disorder and then treat any of the conditions identified. Non-pharmacological and pharmacological methods can be used to treat delirium; however, the first area for intervention is prevention.

Preventative strategies are recommended to reduce the incidence of delirium. It may help patients to stay oriented if clocks are placed within their field of vision, shades are open during the day and lights are turned off at night, and disruptions to sleep are minimised. In one study of such strategies, researchers randomly assigned 852 elderly hospitalised patients to receive either standard medical care alone or along with an intervention designed to prevent delirium. This intervention addressed common risk factors for delirium within the hospital setting, such as sleep deprivation, immobility, dehydration, and visual and hearing impairment. Among other strategies, medical tests and rounds were scheduled later in the morning to avoid waking patients, patients were helped to resume walking soon after surgery, nursing staff made sure that patients stayed hydrated and consumed enough calories and patients' glasses and hearing aids were returned as soon as possible after medical procedures. Patients who received the intervention were less likely to develop delirium and those who did develop delirium recovered more quickly compared to patients who received standard medical care (Inouye et al., 1999).

If an individual is identified as having a delirium; non-pharmacological strategies are the first line of treatment. This includes normalising sleep patterns, avoiding restraints, reorientation and reassurance strategies, relaxation techniques, preventing complications, ensuring adequate hydration, providing clear instructions, minimising hearing or visual impairments (e.g., hearing aids/glasses), limiting room and staff changes, and ensuring pain relief is adequate, to name a few. Pharmacological treatments for delirium (e.g., antipsychotic medications) should only be considered if the individual's behaviour or emotional disturbance is so severe that it threatens the person's or others safety, will interfere with medical care and when non-pharmacological approaches failed (DOHS, 2006; Fong et al., 2009).

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## SUMMARY

### **11.1 Differentiate common misconceptions from established findings about age-related changes and discuss issues involved in conducting research on ageing.**

As the number of older people increases, the demand for mental health professionals specialising in aged care will rise. At present, very few psychologists in Australia report specialising in this area. It is important to be well informed about ageing and age-related issues moving forward. As we age, we tend to become more effective at regulating emotions, to downplay medical symptoms and to focus on core relationships. Negative stereotypes about ageing are sometimes held even by those who are older and the effects of such negative self-beliefs can be quite damaging. There are some challenges associated with becoming older including declining health, changes in sleep and the death of loved ones. There are many changes that the individual needs to cope with and adjust to. In addition, as the number of chronic health conditions increases, polypharmacy may occur.

In research on ageing, it is difficult to disentangle age effects, cohort effects and time-of-measurement effects. Cross-sectional studies do not help distinguish age and cohort effects. Longitudinal studies provide more clarity about age and cohort effects, but the validity of findings can be challenged by attrition. One form of attrition, selective mortality, is particularly important to consider in studies of ageing.

### **11.2 Describe the prevalence of psychological disorders in the elderly and issues involved in estimating the prevalence.**

When older adults present with psychological disorders, it is critically important to evaluate potential medical causes. The elderly are particularly susceptible to the negative effects of medical conditions and medications, and these effects may mimic psychological conditions.

National surveys suggest lower rates of psychological disorders among the elderly compared to other age groups. Although some methodological issues (cohort effects, selective mortality and lack of disclosure) might explain part of this effect, it is also possible that some people become more psychologically healthy as they age.

### **11.3. Explain the symptoms, aetiology and treatment of cognitive disorders in the elderly.**

Dementia describes a range of clinical syndromes characterised by progressive deterioration of cognitive abilities to the point that functioning becomes impaired. There are many different types of dementia and the nature and progression of symptoms varies with each type. *Mild cognitive impairment* is the term used to describe cognitive decline before functional impairment is present.

Alzheimer's disease is characterised by early loss of memory for recent events, and plaques and tangles in the brain. Risk of developing the disease is higher among those with at least one *ApoE-4* allele. Immune and inflammation processes may increase vulnerability to Alzheimer's disease. Lifestyle and psychological factors (e.g. depression, exercise and cognitive engagement) also appear to play a part. Frontotemporal dementia (FTD) is characterised by neuronal deterioration in the frontal and temporal lobes. There are behavioural and language variants of FTD. The primary symptoms of bvFTD include marked changes in social and emotional behaviour. Vascular dementia often occurs after a stroke. The symptoms of vascular dementia depend on the brain regions that are affected by the cerebrovascular disease. Dementia with Lewy bodies is characterised by visual hallucinations, fluctuations in cognitive functioning and sensitivity to side effects of antipsychotic medications.

There is no cure for dementia. Cholinesterase inhibitors are the major medical treatments for dementia, but these medications offer modest effects. Other recommended treatments include exercise, general strategies to improve memory and behavioural interventions.

Delirium is a clinical diagnosis. It has an acute onset (hours to days) and is characterised by fluctuating consciousness, attention and cognition, occurring in the context of a medical condition or substance intoxication or withdrawal. There are different types of delirium including hyperactive, hypoactive and mixed delirium. Treatment includes identifying and treating the underlying

cause, preventative strategies and non-pharmacological strategies (e.g., normalising sleep patterns, reorientation, minimising hearing or visual impairments). Pharmacological treatments such as antipsychotics should be considered if the person's behaviour or emotions impact on his or her safety or the safety of others, or interfere with medical treatment. Non-pharmacological treatment should always be trialled before medication.

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## KEY TERMS

**age effects** the consequences of being a given chronological age

**Alzheimer's disease** a dementia involving a progressive atrophy of cortical tissue and marked by memory impairment, intellectual deterioration and loss of motivation

**cohort effects** the consequences of having been born in a given year and having grown up during a particular time period with its own unique pressures, problems, challenges and opportunities

**cognitive reserve** the idea that some people may be able to compensate for impacts of disease in the brain by using alternative brain networks or cognitive strategies such that cognitive symptoms are less pronounced

**delirium** a state of great mental confusion in which consciousness is clouded, attention cannot be sustained and the stream of thought and speech is incoherent. The person is probably disoriented, emotionally erratic, restless or lethargic and often has illusions, delusions and hallucinations

**dementia** deterioration of mental faculties — memory, judgement, abstract thought, control of impulses, intellectual ability — that impairs social and occupational functioning, and eventually changes the personality

**dementia with Lewy bodies (DLB)** a form of dementia that often co-occurs with Parkinson's disease; characterised by shuffling gait, fluctuating attention and cognition, and hallucinations and delusions

**disorientation** a state of mental confusion with respect to time; place; and identity of self, other persons and objects

**frontotemporal dementia (FTD)** dementia that begins typically in the mid- to late 50s, characterised by deficits in executive functions such as planning, problem solving and goal-directed behaviour as well as recognition and comprehension of emotions in others

**mild cognitive impairment** cognitive decline, based on self/other reports and objective tests, which do not impact on function/daily activities

**neurofibrillary tangles** abnormal protein filaments present in the cell bodies of brain cells in patients with Alzheimer's disease

**plaques** small, round areas composed of remnants of lost neurons and beta-amyloid, a waxy protein deposit; present in the brains of patients with Alzheimer's disease

**polypharmacy** prescription of multiple medications to an individual, traditionally defined as the use of five or more medications

**selective mortality** the tendency for less healthy individuals to die more quickly, which leads to biased samples in long-term follow-up studies

**social selectivity** the late-life shift in interest away from seeking new social interactions and towards cultivating those few social relationships that matter most, such as with family and close friends

**time-of-measurement effects** a possible confound in longitudinal studies whereby conditions at a particular point in time can have a specific effect on a variable that is being studied over time

**vascular dementia** a form of dementia caused by cerebrovascular disease, most commonly occurring after strokes. Because the areas of the brain affected by disease can vary, the symptoms of vascular dementia vary as well

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## WEBSITES

1. Australian Psychological Society (APS) provides information about clinical practice as a psychologist and guidelines for working with older adults. ([www.psychology.org.au/public-interest/ageing](http://www.psychology.org.au/public-interest/ageing))
2. Alzheimer's Australia provides information, education and support to individuals diagnosed with dementia and their caregivers. ([www.fightdementia.org.au](http://www.fightdementia.org.au))
3. Alzheimer's New Zealand provides information about dementia, dementia care and research. ([www.alzheimers.org.nz](http://www.alzheimers.org.nz))
4. Wicking Dementia Research and Education Centre provides a free online course on understanding dementia. ([www.utas.edu.au/wicking/understanding-dementia](http://www.utas.edu.au/wicking/understanding-dementia))

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## REFERENCES

- Acton, G. J., & Kang, J. (2001). Interventions to reduce the burden of caregiving for an adult with dementia: A meta-analysis. *Research in Nursing and Health*, 24, 349–360.
- Ahmed, S., Leurent, B., & Sampson, E. L. (2014). Risk factors for incident delirium among older people in acute hospital medical units: a systematic review and meta-analysis. *Age and Ageing*, 43(3), 326–333. doi:10.1093/ageing/afu022
- Albert, M. S., Dekosky, S. T., Dickson, D., Dubois, B., Feldman, H. H., Fox, N. C., et al. (2011). The diagnosis of mild cognitive impairment due to Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*, 7, 270–279.
- Alpert, P. T., & Gatlin, T. (2015). Polypharmacy in older adults. *Home Healthcare Now November/December*, 33(10), 524–529.
- Alwahhabi, F. (2003). Anxiety symptoms and generalized anxiety disorder in the elderly: A review. *Harvard Review of Psychiatry*, 11, 180–193.
- American Psychiatric Association. (2004). Practice guidelines for the treatment of patients with schizophrenia (2nd ed.) Available online at <http://www.psych.org>.
- American Psychiatric Association (APA). (2013). *Diagnostic and statistical manual of mental disorders* (5th ed.). Arlington, VA: American Psychiatric Publishing.
- Ancoli, I. S., Kripke, D. F., Klauber, M. R., Fell, R., Stepnowsky, C., Estline, E., et al. (1996). Morbidity, mortality and sleep-disordered breathing in community dwelling elderly. *Sleep: Journal of Sleep Research and Sleep Medicine*, 19, 277–282.
- Angst, F., Stassen, H. H., Clayton, P. J., & Angst, J. (2002). Mortality of patients with mood disorders: Follow-up over 34–38 years. *Journal of Affective Disorders*, 68, 167–181.
- Anguera, J. A., Boccanfuso, J., Rintoul, J. L., Al-Hashimi, O., Faraji, F., Janowich, J., et al. (2013). Video game training enhances cognitive control in older adults. *Nature*, 501, 97–101.
- Ascher, E. A., Sturm, V. E., Seider, B. H., Holley, S. R., Miller, B. L., & Levenson, R. W. (2010). Relationship satisfaction and emotional language in frontotemporal dementia and Alzheimer's disease patients and spousal caregivers. *Alzheimer's Disease and Associated Disorders*, 24, 49–55.
- Australian Bureau of Statistics (ABS). (2007). *National survey of mental health and wellbeing: summary of results*, cat. no. 4326.0. Canberra: ABS.
- Australian Bureau of Statistics (ABS). (2012). *Causes of death Australia, 2012*. Canberra: ABS.
- Australian Bureau of Statistics (ABS). (2013). *Population projections, Australia, 2012 (base) to 2101*, cat. no. 3222.0. Canberra: ABS.
- Australian Bureau of Statistics (ABS). (2014a). *Australian demographic statistics*. Canberra: ABS.
- Australian Bureau of Statistics (ABS). (2014b). *Australian historical population statistics*, cat. no.3105.0.65.001. Canberra: ABS.
- Australian Institute of Health and Welfare (AIHW). (2012). *Dementia in Australia*, cat. no. AGE 70. Canberra: AIHW.
- Australian Psychological Society (APS). (2014). *Ethical guidelines for working with older adults*. Retrieved from [www.psychology.org.au/public-interest/ageing/](http://www.psychology.org.au/public-interest/ageing/)
- Balsis, S., Carpenter, B. D., & Storandt, M. (2005). Personality change precedes clinical diagnosis of dementia of the Alzheimer type. *Journal of Gerontology*, 60B, 98–101.
- Balsis, S., Gleason, M. E., Woods, C. M., & Oltmanns, T. F. (2007). An item response theory analysis of DSM-IV personality disorder criteria across younger and older age groups. *Psychology and Aging*, 22, 171–185.
- Birks, J. (2006). Cholinesterase inhibitors for Alzheimer's disease. *Cochrane Database of Systematic Reviews* CD005593.
- Bischof, J., Busse, A., & Angermeyer, M. C. (2002). Mild cognitive impairment: A review of prevalence, incidence and outcome according to current approaches. *Acta Psychiatrica Scandinavica*, 106, 403–414.
- Bookheimer, S., & Burggren, A. (2009). APOE-4 genotype and neurophysiological vulnerability to Alzheimer's and cognitive aging. *Annual Review of Clinical Psychology*, 5, 343–362.

- Brookmeyer, R., Corrada, M. M., Curriero, F. C., & Kawas, C. (2002). Survival following a diagnosis of alzheimer disease. *Archives of Neurology*, 59(11), 1764–1767. doi:10.1001/archneur.59.11.1764
- Buchanan, J. A., Christenson, A., Houlihan, D., & Ostrom, C. (2011). The role of behavior analysis in the rehabilitation of persons with dementia. *Behavior Therapy*, 42, 9–21.
- Camus, V., Burtin, B., Simeone, I., Schwed, P., Gonthier, R., & Dubos, G. (2000). Factor analysis supports the evidence of existing hyperactive and hypoactive subtypes of delirium. *International Journal of Geriatric Psychiatry*, 15, 313–316.
- Carstensen, L. L. (1996). Evidence for a life-span theory of socioemotional selectivity. *Current Directions in Psychological Science*, 4, 151–156.
- Carstensen, L. L., Pasupathi, M., Mayr, U., & Nesselroade, J. R. (2000). Emotional experience in everyday life across the adult life span. *Journal of Personality and Social Psychology*, 79(4), 644–655.
- Caselli, R. J., & Yaari, R. (2008). Medical management of frontotemporal dementia. *American Journal of Alzheimer's Disease and Other Dementias*, 22, 489–498.
- Charles, S. T., Reynolds, C. A., & Gatz, M. (2001). Age-related differences and change in positive and negative affect over 23 years. *Journal of Personality and Social Psychology*, 80(1), 136–151. doi:10.1037/0022-3514.80.1.136
- Cole, M. G. (2004). Delirium in elderly patients. *American Journal of Geriatric Psychiatry*, 12, 7–21.
- Crowley, K. (2011). Sleep and sleep disorders in older adults. *Neuropsychology Review*, 21(1), 41–53. doi:10.1007/s11065-010-9154-6
- Cruts, M., Gijssels, I., van der Zee, J., Engelborghs, S., Wils, H., Pirici, D., et al. (2006). Null mutations in progranulin cause ubiquitin-positive frontotemporal dementia linked to chromosome 17q21. *Nature*, 442, 920–924.
- Curtis, L. H., Ostbye, T., Sendersky, V., Hutchison, S., Dans, P. E., Wright, A., et al. (2004). Inappropriate prescribing for elderly Americans in a large outpatient population. *Archives of Internal Medicine*, 164, 1621–1625.
- Davison, T. E., McCabe, M., & Mellor, D. (2009). An examination of the “gold standard” diagnosis of major depression in aged-care settings. *American Journal of Geriatric Psychiatry*, 17, 359–367.
- Department of Human Services (DOHS). (2006). *Clinical practice guidelines for the management of delirium in older people*. Melbourne, Victoria: Victorian Government Department of Human Services.
- Diniz, B. S., Butters, M. A., Albert, S. M., Dew, M. A., & Reynolds, C. F., III. (2013). Late-life depression and risk of vascular dementia and Alzheimer's disease: Systematic review and meta-analysis of community-based cohort studies. *The British Journal of Psychiatry: The Journal of Mental Science*, 202, 329–335.
- Dugger, M., Ritchie, B. G., Ball, J., Pasyuk, E., Adams, G., Anciant, E., et al. (2002). Eta photoproduction on the proton for photon energies from 0.75 to 1.95 GeV. *Physical Review Letters*, 89, 222002.
- Dura, J. R., Stukenberg, K. W., & Kiecolt-Glaser, J. K. (1991). Anxiety and depressive disorders in adult children caring for demented parents. *Psychology and Aging*, 6, 467–473.
- Ferreira, S. T., Clarke, J. R., Bomfim, T. R., & De Felice, F. G. (2014). Inflammation, defective insulin signaling, and neuronal dysfunction in Alzheimer's disease. *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*, 10, S76–83.
- Fetveit, A. (2009). Late-life insomnia: A review. *Geriatrics and Gerontology International*, 9, 220–234.
- Fillmore, K. M. (1987). Prevalence, incidence and chronicity of drinking patterns and problems among men as a function of age: A longitudinal and cohort analysis. *British Journal of Addiction*, 82, 77–83.
- Fisher, J. E. (2011). Understanding behavioral health in late life: Why age matters. *Behavior Therapy*, 42, 143–149.
- Fisher, J. E., & Noll, J. P. (1996). Anxiety disorders. In L. L. Carstensen, B. A. Edelstein & L. Dornbrand (Eds.), *The practical handbook of clinical gerontology* (pp. 304–323). Thousand Oaks, CA: Sage.
- Fleminger, S., Oliver, D. L., Lovestone, S., Rabe-Hesketh, S., & Giora, A. (2003). Head injury as a risk factor for Alzheimer's disease: the evidence 10 years on; a partial replication. *Journal of Neurology, Neurosurgery & Psychiatry*, 74(7), 857–862. doi:10.1136/jnnp.74.7.857
- Fong, T. G., Tulebaev, S. R., & Inouye, S. K. (2009). Delirium in elderly adults: diagnosis, prevention and treatment. *Nature reviews. Neurology*, 5(4), 210–220. doi:10.1038/nrneurol.2009.24
- Food and Drug Administration. (2005). Deaths with antipsychotics in elderly patients with behavioral disturbances. Public Health Advisory.
- Forbes, D., Thiessen, E. J., Blake, C. M., Forbes, S. C., & Forbes, S. (2013). Exercise programs for people with dementia. *The Cochrane Database of Systematic Reviews*, 12, CD006489.
- Frederickson, B. L., & Carstensen, L. L. (1990). Choosing social partners: How old age and anticipated endings make people more selective. *Psychology and Aging*, 5, 335–347.
- Frojd, K., Hakansson, A., Karlsson, I., & Molarius, A. (2003). Deceased, disabled or depressed—A population-based 6-year followup study of elderly people with depression. *Social Psychiatry and Psychiatric Epidemiology*, 38, 557–562.
- Gallo, J. J., & Lebowitz, B. D. (1999). The epidemiology of common late-life mental disorders in the community: Themes for the new century. *Psychiatric Services*, 50, 1158–1166.
- Gandy, S. (2014). Alzheimer's disease: new data highlight nonneuronal cell types and the necessity for presymptomatic prevention strategies. *Biological Psychiatry*, 75, 553–557.
- Ganguli, M., Du, Y., Dodge, H. H., Ratcliff, G. G., & Chang, C.-C. H. (2006). Depressive symptoms and cognitive decline in late life: A prospective epidemiological study. *Archives of General Psychiatry*, 63, 153–160.

- Garand, L., Buckwalter, K. C., Lubaroff, D., Tripp-Reimer, T., Frantz, R. A., & Ansley, T. N. (2002). A pilot study of immune and mood outcomes of a community-based intervention for dementia caregivers: The PLST intervention. *Archives of Psychiatric Nursing*, 16, 156–167.
- Geser, F., Wenning, G. K., Poewe, W., & McKeith, I. (2005). How to diagnose dementia with Lewy bodies: state of the art. *Movement Disorders*, 20(12), S11–20. doi:10.1002/mds.20535
- Gitlin, L. N., Winter, L., Burke, J., Chernett, N., Dennis, M. P., & Hauck, W. W. (2008). Tailored activities to manage neuropsychiatric behaviours in persons with dementia and reduce caregiver burden: A randomized pilot study. *The American Journal of Geriatric Psychiatry*, 16(3), 229–239. doi:10.1097/JGP.0b013e318160da72
- Goodkind, M. S., Gyurak, A., McCarthy, M., Miller, B. L., & Levenson, R. W. (2010). Emotion regulation deficits in frontotemporal lobar degeneration and Alzheimer's disease. *Psychological Aging*, 25, 30–37.
- Gorelick, P. B. (2010). Role of inflammation in cognitive impairment: results of observational epidemiological studies and clinical trials. *Annals of the New York Academy of Sciences*, 1207, 155–162.
- Gorno-Tempini, M. L., Hillis, A. E., Weintraub, S., Kertesz, A., Mendez, M., Cappa, S. F., ... Grossman, M. (2011). Classification of primary progressive aphasia and its variants. *Neurology*, 76(11), 1006–1014. doi:10.1212/WNL.0b013e31821103e6
- Goveas, J. S., Espeland, M. A., Woods, N. F., Wassertheil-Smoller, S., & Kotchen, J. M. (2011). Depressive symptoms and incidence of mild cognitive impairment and probable dementia in elderly women: The Women's Health Initiative memory study. *Journal of the American Geriatrics Society*, 59, 57–66.
- Grattan, M. (2003). *Hazel Hawke: I have Alzheimer's*. Retrieved from www.theage.com.au/articles/2003/11/02/1067708071324.html
- Gum, A. M., King-Kallimanis, B., & Kohn, R. (2009). Prevalence of mood, anxiety, and substance-abuse disorders for older Americans in the National Comorbidity Survey-Replication. *American Journal of Psychiatry*, 17, 769–781.
- Hamer, M., & Chida, Y. (2009). Physical activity and risk of neurodegenerative disease: a systematic review of prospective evidence. *Psychological Medicine*, 39, 3–11.
- Hausdorff, J. M., Levy, B. R., & Wei, J. Y. (1999). The power of ageism on physical function of older persons: Reversibility of age-related gait changes. *Journal of the American Geriatrics Society*, 47, 1346–1349.
- Head, D., Bugg, J. M., Goate, A. M., Fagan, A. M., Mintun, M. A., Benzinger, T., et al. (2012). Exercise engagement as a moderator of the effects of APOE genotype on amyloid deposition. *Archives of neurology*, 69, 636–643.
- Hertzog, C., Kramer, A., Wilson, R. S., & Lindenberger, U. (2009). Enrichment effects on adult cognitive development: Can the functional capacity of older adults be preserved and enhanced? *Psychological Science in the Public Interest*, 9.
- Heyn, P., Abreu, B. C., & Ottenbacher, K. J. (2004). Meta-analysis: The effects of exercise training on elderly persons with cognitive impairment and dementia: A meta-analysis. *Archives of Physical Medicine and Rehabilitation*, 85, 1694–1704.
- Hill, N. T. M., Mowszowski, L., Naismith, S. L., Chadwick, V. L., Valenzuela, M., & Lampit, A. (2016). Computerized cognitive training in older adults with Mild Cognitive Impairment or dementia: A systematic review and meta-analysis. *American Journal of Psychiatry*. doi:10.1176/appi.ajp.2016.16030360
- Hodges, J. R., Patterson, K., Oxbury, S., & Funnell, E. (1992). Semantic dementia. *Brain*, 115(6), 1783–1806.
- Holmes, C., Boche, D., Wilkinson, D., Yadegarfar, G., Hopkins, V., Bayer, A., et al. (2008). Long-term effects of Abeta42 immunization in Alzheimer's disease: Follow-up of a randomised, placebo-controlled phase I trial. *The Lancet*, 372, 216–223.
- Howard, R., McShane, R., Lindesay, J., Ritchie, C., Baldwin, A., Barber, R., et al. (2012). Donepezil and memantine for moderate-to-severe Alzheimer's disease. *New England Journal of Medicine*, 366, 893–903.
- Howard, R., Rabins, P. V., Seeman, M. V., & Jeste, D. V. (2000). Late-onset schizophrenia and very-late-onset schizophrenia-like psychosis: an international consensus. The International Late-Onset Schizophrenia Group. *American Journal of Psychiatry*, 157, 172–178.
- Hu, W. T., Seelaar, H., Josephs, K. A., Knopman, D. S., Boeve, B. F., Sorenson, E. J., et al. (2009). Survival profiles of patients with frontotemporal dementia and motor neuron disease. *Archives of Neurology*, 66, 1359–1364.
- Inouye, S. K., Bogardus, S. T., Jr., Charpentier, P. A., Leo-Summers, L., Acampora, D., Holford, T. R., et al. (1999). A multicomponent intervention to prevent delirium in hospitalized older patients. *New England Journal of Medicine*, 340, 669–676.
- Isaacowitz, D. M. (2012). Mood regulation in real time: Age differences in the role of looking. *Current Directions in Psychological Science*, 21, 237–242.
- Jack, C. R., Jr., Albert, M. S., Knopman, D. S., McKhann, G. M., Sperling, R. A., Carrillo, M. C., et al. (2011). Introduction to the recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*, 7, 257–262.
- Jackson, J. C., Gordon, S. M., Hart, R. P., Hopkins, R. O., & Ely, E. W. (2004). The association between delirium and cognitive decline: A review of the empirical literature. *Neuropsychology Review*, 14, 87–98.
- Jak, A. J., Bondi, M. W., Delano-Wood, L., Wierenga, C., Corey-Bloom, J., Salmon, D., et al. (2009). Quantification of five neuropsychological approaches to defining mild cognitive impairment. *American Journal of Geriatric Psychiatry*, 17, 368–375.
- Jones, L., Holmans, P. A., Hamshere, M. L., Harold, D., Moskvina, V., Ivanov, D., ... Sims, R. (2010). Genetic evidence implicates the immune system and cholesterol metabolism in the aetiology of Alzheimer's disease. *PLoS one*, 5(11), e13950.
- Josephs, K. A. (2008). Frontotemporal dementia and related disorders: Deciphering the enigma. *Annals of Neurology*, 64, 4–14.
- Karon, B. P., & VandenBos, G. R. (1998). Schizophrenia and psychosis in elderly populations. In I. H. Nordhus, G. R. VandenBos, S. Berg & P. Fromholt (Eds.), *Clinical Geropsychology* (pp. 219–227). Washington, DC: American Psychological Association.
- Katz, I. R., Parmelee, P., & Brubaker, K. (1991). Toxic and metabolic encephalopathies in long-term care patients. *International Psychogeriatrics*, 3, 337–347.

- Kessler, L. (August 22, 2004). Dancing with Rose: A strangely beautiful encounter with Alzheimer's patients provides insights that challenge the way we view the disease. *Los Angeles Times Magazine*.
- Kiecolt-Glaser, J. K., & Glaser, R. (2001). Stress and immunity: Age enhances the risks. *Current Directions in Psychological Science*, 10, 18–21.
- Kiecolt-Glaser, J. K., & Glaser, R. (2002). Depression and immune function: Central pathways to morbidity and mortality. *Journal of Psychosomatic Research*, 53, 873–876.
- Kiecolt-Glaser, J. K., Dura, J. R., Speicher, C. E., & Trask, O. (1991). Spousal caregivers of dementia victims: Longitudinal changes in immunity and health. *Psychosomatic Medicine*, 54, 345–362.
- Kisley, M. A., Wood, S., & Burrows, C. L. (2007). Looking at the sunny side of life: Age-related change in an event-related potential measure of the negativity bias. *Psychological Science*, 18, 838.
- Klunk, W. E., Engler, H., Nordberg, A., Wang, Y., Blomqvist, G., & Holt, D. P. (2004). Imaging brain amyloid in Alzheimer's disease with Pittsburgh Compound-B. *Annals of Neurology*, 55, 306–319.
- Knight, B. G. (1996). *Psychotherapy with older adults* (2nd ed.). Thousand Oaks, CA: Sage.
- Koder, D. A., & Helmes, E. (2008). The current status of clinical geropsychology in Australia: A survey of practising psychologists. *Australian Psychologist*, 43(1), 22–26. doi:10.1080/00050060701710967
- Kubzansky, L. D. (2007). Sick at heart: The pathophysiology of negative emotions. *Cleveland Clinic Journal of Medicine*, 74 Suppl 1, S67–72.
- Langa, K. M., Larson, E. B., Karlawish, J. H., Cutler, D. M., Kabeto, M. U., Kim, S. Y., et al. (2008). Trends in the prevalence and mortality of cognitive impairment in the United States: Is there evidence of a compression of cognitive morbidity? *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*, 4, 134–144.
- Lauril, J. V., Pitkälä, K. H., Strandberg, T. E., & Tilvis, R. S. (2004). Detection and documentation of dementia and delirium in acute geriatric wards. *General Hospital Psychiatry*, 26, 31–35.
- Levenson, R. W., & Miller, B. L. (2007). Loss of cells—Loss of self: Frontotemporal lobar degeneration and human emotion. *Current Directions in Psychological Science*, 16, 289–294.
- Levenson, R. W., Carstensen, L. L., & Gottman, J. M. (1994). Influence of age and gender on affect, physiology, and their interrelations: A study of long-term marriages. *Journal of Personality and Social Psychology*, 67, 56–68.
- Levy, B. R. (1996). Improving memory in old age through implicit self-stereotyping. *Journal of Personality and Social Psychology*, 71, 1092–1107.
- Levy, B. R. (2003). Mind matters: Cognitive and physical effects of aging self-stereotypes. *Journal of Gerontology: Psychological Sciences*, 58B, 203–211.
- Levy, B. R., Slade, M. D., & Kasl, S. V. (2002). Longitudinal benefit of positive self-perceptions of aging on functional health. *Journal of Gerontology: Psychological Sciences*, 57B, 409–417.
- Lieb, W., Beiser, A. S., Vasan, R. S., Tan, Z. S., Au, R., Harris, T. B., et al. (2009). Association of plasma leptin levels with incident Alzheimer disease and MRI measures of brain aging. *Journal of the American Medical Association*, 302, 2565–2572.
- Livingston, G., Johnston, K., Katona, C., Paton, J., & Lyketsos, C. G. (2005). Systematic review of psychological approaches to the management of neuropsychiatric symptoms of dementia. *American Journal of Psychiatry*, 162, 1996–2021.
- Lockwood, K. A., Alexopoulos, G. S., Kakuma, T., & van Gorp, W. G. (2000). Subtypes of cognitive impairment in depressed older adults. *American Journal of Geriatric Psychiatry*, 8, 201–208.
- Logsdon, R. G., McCurry, S. M., & Teri, L. (2007). Evidence-based psychological treatments for disruptive behaviors in individuals with dementia. *Psychology and Aging*, 22, 28–36.
- Lonergan, E., Britton, A. M., & Luxenberg, J. (2007). Antipsychotics for delirium. *Cochrane Database of Systematic Reviews*, Issue 2, CD005594.
- Lopez, O. L., Kuller, L. H., Becker, J. T., Jagust, W. J., DeKosky, S. T., Fitzpatrick, A., . . . Carlson, M. (2005). Classification of vascular dementia in the Cardiovascular Health Study Cognition Study. *Neurology*, 64(9), 1539–1547.
- Lyketsos, C., & Liang, B. (1999). Diagnosis and management of delirium in the elderly. *Hospital Physician*, 35(6), 34–51.
- Mackenzie, I. R., Neumann, M., Bigio, E. H., Cairns, N. J., Alafuzoff, I., Kril, J., et al. (2009). Nomenclature for neuropathologic subtypes of frontotemporal lobar degeneration: consensus recommendations. *Acta Neuropathologica*, 117, 15–18.
- Maidment, I., Fox, C., & Boustani, M. (2006). Cholinesterase inhibitors for Parkinson's disease dementia. *Cochrane Database of Systematic Reviews*, Issue 1, CD004747.
- Marcantonio, E. R., Flacker, J. M., Wright, R. J., & Resnick, N. M. (2001). Reducing delirium after hip fracture: A randomized trial. *Journal of the American Geriatrics Society*, 49, 516–522.
- Mather, M., & Carstensen, L. L. (2005). ageing and motivated cognition: the positivity effect in attention and memory. *Trends in Cognitive Sciences*, 9(10), 496–502. doi:10.1016/j.tics.2005.08.005
- Mather, M., Canli, T., English, T., Whitfield, S., Wais, P., Ochsner, K., et al. (2004). Amygdala responses to emotionally valenced stimuli in older and younger adults. *Psychological Science*, 15, 259–263.
- McCusker, J., Cole, M., & Abrahamowicz, M. (2002). Delirium predicts 12-month mortality. *Archives of Internal Medicine*, 162, 457–463.
- McKeith, I. G. (2006). Consensus guidelines for the clinical and pathologic diagnosis of dementia with Lewy bodies (DLB): report of the Consortium on DLB International Workshop. *Journal of Alzheimers Disease*, 9(3 Suppl), 417–423.
- McKeith, I. G., Dickson, D. W., Lowe, J., Emre, M., O'Brien, J. T., Feldman, H., . . . Yamada, M. (2005). Diagnosis and management of dementia with Lewy bodies: third report of the DLB Consortium. *Neurology*, 65(12), 1863–1872. doi:10.1212/01.wnl.0000187889.17253.b1

- McKeith, I. G., Galasko, D., Kosaka, K., Perry, E. K., Dickson, D. W., Hansen, L. A., . . . Perry, R. H. (1996). Consensus guidelines for the clinical and pathologic diagnosis of dementia with Lewy bodies (DLB): report of the consortium on DLB international workshop. *Neurology*, 47(5), 1113–1124.
- McKhann, G. M., Knopman, D. S., Chertkow, H., Hyman, B. T., Jack, C. R., Jr., Kawas, C. H., et al. (2011). The diagnosis of dementia due to Alzheimer's disease: Recommendations from the National Institute on Aging–Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's & Dementia: The Journal of the Alzheimer's Association*, 7, 263–269.
- Meagher, D. J. (2007). Phenomenology of delirium: Assessment of 100 adult cases using standardised measures. *British Journal of Psychiatry*, 190, 135–141.
- Mendez, M. F., & Shapira, J. S. (2008). The spectrum of recurrent thoughts and behaviors in frontotemporal dementia. *CNS Spectrums*, 13, 202–208.
- Mendez, M. F., Lauterbach, E. C., & Sampson, S. M. (2008). An evidence-based review of the psychopathology of frontotemporal dementia: A report of the ANPA Committee on Research. *Journal of Neuropsychiatry and Clinical Neurosciences*, 20, 130–149.
- Mesulam, M. M. (2003). Primary progressive aphasia- a language-based dementia. *New England Journal of Medicine*, 349(16), 1535–1542. doi:10.1056/NEJMra022435
- Mesulam, M. M. (2013). Primary progressive aphasia: A dementia of the language network. *Dementia & Neuropsychologia*, 7(1), 2–9.
- Miller, B. L., Ikonte, C., Ponton, M., & Levy, M. (1997). A study of the Lund Manchester research criteria for frontotemporal dementia: Clinical and single-photon emission CT correlations. *Neurology*, 48, 937–942.
- Mittelman, M. S., Brodaty, H., Wallen, A. S., & Burns, A. (2008). A three-country randomized controlled trial of a psychosocial intervention for caregivers combined with pharmacological treatment for patients with Alzheimer disease: Effects on caregiver depression. *American Journal of Geriatric Psychiatry*, 16, 893–904.
- Modrego, P. J. (2010). Depression in Alzheimer's disease: Pathophysiology, diagnosis, and treatment. *Journal of Alzheimer's Disease*, 21, 1077–1087.
- Moorhouse, P., & Rockwood, K. (2008). Vascular cognitive impairment: current concepts and clinical developments. *Lancet Neurol*, 7(3), 246–255. doi:10.1016/s1474-4422(08)70040-1
- Moroney, J. T., Tang, M. X., Berglund, L., Small, S., Merchant, C., Bell, K., et al. (1999). Low-density lipoprotein cholesterol and the risk of dementia with stroke. *Journal of American Medical Association*, 282, 254–260.
- National Academy on an Aging Society. (1999). *Challenges for the 21st century: Chronic and disabling conditions*. Retrieved from [www.agingociety.org/agingociety/publications/chronic/index.html](http://www.agingociety.org/agingociety/publications/chronic/index.html).
- Norton, M. C., Skoog, I., Toone, L., Corcoran, C., Tschanz, J. T., Lisota, R. D., et al. (2006). Three-year incidence of first-onset depressive syndrome in a population sample of older adults: The Cache County Study. *American Journal of Geriatric Psychiatry*, 14, 237–245.
- O'Connor, C. M., Clemson, L., Brodaty, H., Jeon, Y. H., Mioshi, E., & Gitlin, L. N. (2014). Use of the Tailored Activities Program to reduce neuropsychiatric behaviours in dementia: an Australian protocol for a randomized trial to evaluate its effectiveness. *International Psychogeriatrics*, 26(5), 857–869. doi:10.1017/S1041610214000040
- Oakley Browne, M. A., Wells, J. E., & Scott, K. M. (2006). *Te Rau Hinengaro: the New Zealand mental health survey*. Wellington.
- Ogden, J. A. (2005). Dementia: a family tragedy. In *Fractured minds: a case-study approach to clinical neuropsychology* (2nd ed., pp. 304–327). New York: Oxford University Press.
- Oliveira, A. M. d., Radanovic, M., Mello, P. C. H. d., Buchain, P. C., Vizzotto, A. D. B., Celestino, D. L., . . . Forlenza, O. V. (2015). Nonpharmacological interventions to reduce behavioural and psychological symptoms of dementia: A systematic review. *BioMed Research International*, 2015, 9. doi:10.1155/2015/218980
- Ory, M., Hoffman, R. R., Yee, J. L., Tennstedt, S., & Schulz, R. (1999). Prevalence and impact of caregiving: A detailed comparison between dementia and nondementia caregivers. *The Gerontologist*, 39, 177–185.
- Ozten, E., Hizli, S., Salcini, C., Kagan, G., & Tanridag, O. (2013). Frontotemporal dementia patient with bipolar disorder: a case report. *OA Case Reports*, 2(2), 16–18.
- Pachana, N. A., Helmes, E., & Koder, D. (2006). Guidelines for the provision of psychological services for older adults. *Australian Psychologist*, 41(1), 15–22. doi:10.1080/00050060500391852
- Pendlebury, S. T., & Rothwell, P. M. (2009). Risk of recurrent stroke, other vascular events and dementia after transient ischaemic attack and stroke. *Cerebrovascular Diseases*, 27 Suppl 3, 1–11.
- Petersen, R. C. (2011). Mild cognitive impairment. *New England Journal of Medicine*, 364(23), 2227–2234. doi:10.1056/NEJMcp0910237
- Prechter, G. C., & Shepard, J. W. J. (1990). Sleep and sleep disorders in the elderly. In R. J. Martin (Ed.), *Cardiorespiratory disorders during sleep* (pp. 365–386). Armonk, NY: Futura Publishing Co.
- Pressman, P. S., & Miller, B. L. (2014). Diagnosis and management of behavioral variant frontotemporal dementia. *Biological Psychiatry*, 75, 574–581.
- Prince, M., Bryce, R., Albanese, E., Wimo, A., Ribeiro, W., & Ferri, C. P. (2013). The global prevalence of dementia: A systematic review and metaanalysis. *Alzheimer's & Dementia*, 9(1), 63–75.e62. doi:http://dx.doi.org/10.1016/j.jalz.2012.11.007
- Qato, D. M., Alexander, G. C., Conti, R. M., Johnson, M., Schumm, P., & Lindau, S. T. (2008). Use of prescription and over-the-counter medications and dietary supplements among older adults in the United States. *Journal of the American Medical Association*, 300, 2867–2878.

- Quals, S. H., Segal, D. L., Norman, S., Niederehe, G., & Gallagher-Thompson, D. (2002). Psychologists in practice with older adults: Current patterns, sources of training, and need for continuing education. *Professional Psychology: Research and Practice*, 33, 435–442.
- Rapp, M. A., Schnaider-Beeri, M., Grossman, H. T., Sano, M., Perl, D. P., Purohit, D. P., Gorman, J. M., et al. (2006). Increased hippocampal plaques and tangles in patients with Alzheimer disease with a lifetime history of major depression. *Archives of General Psychiatry*, 63, 161–167.
- Rascovsky, K., Hodges, J. R., Knopman, D., Mendez, M. F., Kramer, J. H., Neuhaus, J., et al. (2011). Sensitivity of revised diagnostic criteria for the behavioural variant of frontotemporal dementia. *Brain: A Journal of Neurology*, 134, 2456–2477.
- Rebok, G. W., Ball, K., Guey, L. T., Jones, R. N., Kim, H.-Y., King, J. W., et al. (2014). Ten-year effects of the advanced cognitive training for Independent and Vital Elderly Cognitive Training Trial on cognition and everyday functioning in older adults. *Journal of the American Geriatrics Society*, 62, 16–24.
- Rieger, D. A., Boyd, J. H., Burke, J. D., & Rae, D. S. (1988). One-month prevalence of mental disorders in the United States: Based on five epidemiologic catchment area sites. *Archives of General Psychiatry*, 45, 977–986.
- Ritchie, K., Norton, J., Mann, A., Carriere, I., & Ancelin, M. L. (2013). Late-onset agoraphobia: General population incidence and evidence for a clinical subtype. *American Journal of Psychiatry*, 170, 790–798.
- Roberts, R., & Knopman, D. S. (2013). Classification and epidemiology of MCI. *Clinics in geriatric medicine*, 29(4). doi:10.1016/j.cger.2013.07.003
- Schaie, K. W., & Hertzog, C. (1982). Longitudinal methods. In B. B. Wolman (Ed.), *Handbook of developmental Ppsychology*. Englewood Cliffs, NJ: Prentice-Hall.
- Seelaar, H., Rohrer, J. D., Pijnenburg, Y. A., Fox, N. C., & van Swieten, J. C. (2011). Clinical, genetic and pathological heterogeneity of frontotemporal dementia: a review. *Journal of Neurology, Neurosurgery and Psychiatry*, 82(5), 476–486. doi:10.1136/jnnp.2010.212225
- Selkoe, D. J. (2002). Alzheimer's disease is a synaptic failure. *Science*, 298, 789–791.
- Selwood, A., Johnson, K., Katona, C., Ilyketos, C., & Livingston, G. (2007). Systematic review of the effect of psychological interventions on family caregivers of people with dementia. *Journal of Affective Disorders*, 101, 75–89.
- Shaper, A. G. (1990). Alcohol and mortality: A review of prospective studies. *British Journal of Addiction*, 85, 837–847.
- Siegler, I. C., & Costa, P. T., Jr. (1985). Health behavior relationships. In J. E. Birren & K. W. Schaie (Eds.), *Handbook of the psychology of aging* (2nd ed.). New York: Van Nostrand-Reinhold.
- Small, B. J., Fratiglioni, L., Viitanen, M., Winblad, B., & Backman, L. (2000). The course of cognitive impairment in preclinical Alzheimer disease. *Archives of Neurology*, 57, 839–844.
- Sofi, F., Valecchi, D., Bacci, D., Abbate, R., Gensini, G. F., Casini, A., et al. (2011). Physical activity and risk of cognitive decline: A meta-analysis of prospective studies. *Journal of Internal Medicine*, 269, 107–117.
- Speechly, C. M., Bridges-Webb, C., & Passmore, E. (2008). The pathway to dementia diagnosis. *The Medical Journal of Australia*, 189(9), 487–489.
- Sperling, R. A., Aisen, P. S., Beckett, L. A., Bennett, D. A., Craft, S., Fagan, A. M., et al. (2011). Toward defining the preclinical stages of Alzheimer's disease: Recommendations from the National Institute on Aging-Alzheimer's Association workgroups on diagnostic guidelines for Alzheimer's disease. *Alzheimer's and Dementia: The Journal of the Alzheimer's Association*, 7, 280–292.
- Subramanyam, A., & Singh, S. (2016). Mild cognitive decline: Concept, types, presentation and management. *Journal of Geriatric Mental Health*, 3, 10.
- Sutphen, C. L., Fagan, A. M., & Holtzman, D. M. (2014). Progress update: Fluid and imaging biomarkers in Alzheimer's disease. *Biological Psychiatry*, 75, 520–526.
- Teper, E., & O'Brien, J. T. (2008). Vascular factors and depression. *International Journal of Geriatric Psychiatry*, 23, 993–1000.
- Teri, L., Gibbons, L. E., McCurry, S. M., Logsdon, R. G., Buchner, D. M., Barlow, W. E., et al. (2003). Exercise plus behavioral management in patients with Alzheimer disease: A randomized controlled trial. *Journal of the American Medical Association*, 290, 2015–2022.
- Terry, R. D. (2006). Alzheimer's disease and the aging brain. *Journal of Geriatric Psychiatry and Neurology*, 19, 125–128.
- Torti, F. M., Gwyther, L. P., Reed, S. D., Friedman, J. Y., & Schulman, K. A. (2004). A multinational review of recent trends and reports in dementia caregiver burden. *Alzheimers Disease and Associated Disorders*, 18, 99–109.
- United Nations, D. o. E. a. S. A., Population Division. (2015). *World population ageing 2015*. New York: United Nations.
- Valenzuela, M. J., & Sachdev, P. (2006). Brain reserve and dementia: A systematic review. *Psychological Medicine*, 36, 441–454.
- Vemuri, P., Lesnick, T. G., Przybelski, S. A., Knopman, D. S., Mielke, M. M., Roberts, R. O., et al. (2014). Association of lifetime intellectual enrichment with cognitive decline in the older population. *Journal of the American Medical Association, Neurology*, 71, 1017–1024.
- Vinkers, D. J., Gussekloo, J., Stek, M. L., Westendorp, R. G. J., & van der Mast, R. C. (2004). Temporal relation between depression and cognitive impairment in old age: Prospective population based study. *British Medical Journal*, 329, 881.
- Vitaliano, P. P., Zhang, J., & Scanlan, J. M. (2003). Is caregiving hazardous to one's physical health? A meta-analysis. *Psychological Bulletin*, 129, 946–972.
- Webster, R., & Holroyd, S. (2000). Prevalence of psychotic symptoms in delirium. *Psychosomatics*, 41, 519–522.
- Wells, C. E., & Duncan, G. W. (1980). *Neurology for psychiatrists*. Philadelphia: F. A. Davis.

- Williams, J. W., Plassman, B. L., Burke, J., Holsinger, T., & Benjamin, S. (2010). Preventing Alzheimer's disease and cognitive decline. *Evidence Report/Technology Assessment No. 193*. Rockville, MD: Duke Evidence-based Practice Center.
- Wilson, R. S., Scherr, P. A., Schneider, J. A., Tang, Y., & Bennett, D. A. (2007). Relation of cognitive activity to risk of developing Alzheimer's disease. *Neurology*, 69, 1191–1920.
- Witlox, J., Eurelings, L. S. M., de Jonghe, J. F. M., Kalisvaart, K. J., Eikelenboom, P., & van Gool, W. A. (2010). Delirium in elderly patients and the risk of postdischarge mortality, institutionalization, and dementia. *Journal of the American Medical Association*, 304, 443–451.
- Yaffe, K., Fiocco, A. J., Lindquist, K., Vittinghoff, E., Simonsick, E. M., Newman, A. B., et al. (2009). Predictors of maintaining cognitive function in older adults. *Neurology*, 72, 2029–2035.
- York Cornwell, E., & Waite, L. J. (2009). Social disconnectedness, perceived isolation, and health among older adults. *Journal of Health and Social Behavior*, 50, 31–48.
- Zarit, S. H. (1980). *Aging and mental disorders: Psychological approaches to assessment and treatment*. New York: Free Press.
- Zarit, S. H., & Zarit, J. M. (2011). *Mental disorders in older adults: Fundamentals of assessment and treatment* (2nd ed.). New York: Guilford Press.
- Zhou, J., & Seeley, W. W. (2014). Network dysfunction in Alzheimer's disease and frontotemporal dementia: Implications for psychiatry. *Biological Psychiatry*, 75, 565–573.

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## CHAPTER 12

# Personality and personality disorders

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 12.1** explain the DSM-5 approach to classifying personality disorders, key concerns with this approach and the DSM-5 alternative approach to diagnosis
  - 12.2** describe commonalities in the risk factors across the personality disorders
  - 12.3** discuss the clinical description and aetiology of the odd/eccentric cluster of DSM-5 personality disorders
  - 12.4** discuss the clinical description and aetiology of the dramatic/erratic cluster of DSM-5 personality disorders
  - 12.5** discuss the clinical description and aetiology of the anxious/fearful cluster of DSM-5 personality disorders
  - 12.6** describe the available psychological treatments of the DSM-5 personality disorders.
-

## OPENING SCENARIO

Maria was single and 26 years old when she was first admitted to a psychiatric hospital. She had been in outpatient treatment with a psychologist for several months when her persistent thoughts of cutting, burning and killing herself led her therapist to conclude that she needed more than outpatient treatment.

Maria's first experience with psychotherapy occurred when she was an adolescent. Her grades declined sharply in Year 11 and her parents suspected she was using drugs. She began to miss curfews and, occasionally, to stay out all night. She often skipped school. Family therapy was started and it seemed to go well at first. Maria was enthusiastic about the therapist and asked for additional private sessions with him.

During individual sessions, Maria revealed she had used drugs extensively and had prostituted herself several times to get drug money. Her relationships with her peers were in chronic turmoil. There was a constant parade of new friends, whom Maria at first thought to be the greatest ever but who soon disappointed her and were cast aside, often in very unpleasant ways.

After several weeks of family therapy, Maria's parents noticed that Maria was angry and abusive towards the therapist. After a few more weeks had passed, Maria refused to attend any more sessions. In a subsequent conversation with the therapist, Maria's father learned that she had behaved seductively towards the therapist during their private sessions and that her changed attitude coincided with his rejection of her advances despite the therapist's attempt to mix firmness with warmth and empathy.

Maria managed to complete high school and enrolled in a local TAFE college, but the old patterns returned. Poor grades, continuing drug use and lack of interest in her studies finally led her to quit in the middle of the first semester of her second year. After leaving TAFE, Maria held a series of low-paying jobs. Most of them didn't last long, as her relationships with co-workers paralleled her relationships with her peers in high school. When Maria started a new job, she would find someone she really liked, but something would come between them and the relationship would end angrily. She was often suspicious of her co-workers and reported that she heard them plotting how to prevent her from getting ahead on the job. She was quick to find hidden meanings in their behaviour, as when she interpreted being the last person asked to sign a birthday card to mean that she was the least-liked person in the office. She indicated that she 'received vibrations' from others and could tell when they really didn't like her even in the absence of any direct evidence.

Maria's frequent mood swings, with periods of depression and extreme irritability, led her to seek therapy several times. But after initial enthusiasm, her relationships with therapists always deteriorated, resulting in premature termination of therapy. By the time of her hospitalisation, she had seen six therapists.

## QUESTIONS

1. What are the main features of Maria's symptomatology?
2. Which one of Maria's symptoms puts her at higher risk in terms of her safety?

## Introduction

What sort of a person are you? A response to such a question might be a simple indication of what is typically meant by *personality*. To define personality, some theories refer to traits such as neuroticism, extraversion, agreeableness, conscientiousness and openness to experience (McCrae & Costa, 1984). These traits are considered persistent over time and pervasive across a wide range of life domains. In other words, personality refers to consistent patterns of thinking, feeling and behaving. It refers to the individual differences in how people perceive, think and relate to their environment and themselves.

There are many theories in psychology that attempt to understand and explain human behaviour and personality, such as psychodynamic, behavioural, humanistic and even biological theories. Personality not only includes temperament and learning, but also contains factors such as one's values, attitudes, expectations, interpersonal interactions, coping strategies and self-perception. As such, personality consists of overt and covert actions and conscious and unconscious processes in which all these elements

interact with each other and the environment. That is why it is preferable to talk about personality as a dynamic process rather than a set of characteristics that remain stable throughout the lifespan. Nevertheless, personality is all the more complicated when it comes to the context of abnormal behaviour and disorders (Millon, 2015). According to the DSM-5, when those patterns or processes are inflexible, self-defeating, and cause distress and dysfunctionality, for example in relationships, they constitute personality disorder.

According to DSM-5, a personality disorder is ‘an enduring pattern of inner experience and behaviour that deviates markedly from expectations of the individual’s culture, is pervasive and inflexible, has onset in adolescence or early adulthood, is stable over time and leads to distress or impairment’ (APA, 2013, p. 645). Although these disorders are all defined by extreme and inflexible characteristics, the 10 **personality disorders** cover a broad range of symptom profiles. As examples of that heterogeneity, paranoid personality disorder is defined by chronic tendencies to be mistrustful and suspicious; anti-social personality disorder by patterns of irresponsibility and callous disregard for the rights of others; and dependent personality disorder by an over-reliance on others.

Our personalities form almost every domain of our lives — the quality of our relationships, managing social networks, our preferred level of activity and career, our approach to tackling everyday problems, and our typical level of wellbeing (Ozer & Benet-Martinez, 2006). Given how many areas of our life are shaped by personality traits, it stands to reason that the extreme and maladaptive traits found in personality disorders create problems in multiple domains. People with personality disorders experience difficulties with their identity and their relationships, and these problems are sustained for years.

In this chapter we begin by considering the DSM-5 approach to classifying personality disorders and the assessment of personality disorders. We will note some concerns about the DSM-5 approach, and will then discuss an alternative system of classification that has been placed in the DSM-5 appendix. After considering these broad issues in the classification of personality disorders, we describe factors that increase the risk of personality disorders in general. Then, we discuss the specific personality disorders, including clinical descriptions and risk factors. We conclude with discussion of the treatment of personality disorders.

## 12.1 The DSM-5 approach to classification

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**LEARNING OUTCOME 12.1** Explain the DSM-5 approach to classifying personality disorders, key concerns with this approach and the DSM-5 alternative approach to diagnosis.

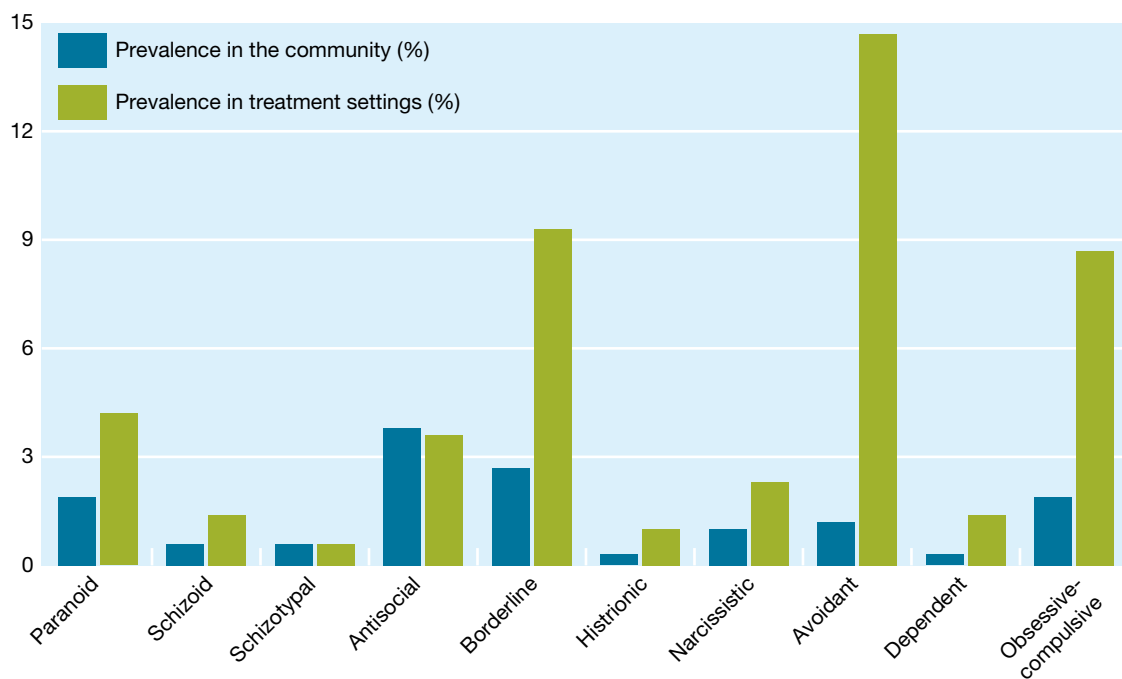
In DSM-5, the 10 different personality disorders are classified in three clusters, reflecting the idea that these disorders are characterised by odd or eccentric behaviour (cluster A); dramatic, emotional or erratic behaviour (cluster B); or anxious or fearful behaviour (cluster C). These clusters form a beneficial organisational framework for our discussions in this chapter. Table 12.1 presents the personality disorders, their key features and their grouping in clusters.

About 1 out of every 10 people meet the diagnostic criteria for a personality disorder (Sansone & Sansone, 2011). With rates this high, it is likely you know people who would meet the diagnostic criteria for a personality disorder.

Personality disorders tend to be more common among people with a psychological disorder such as major depressive disorder or an anxiety disorder. In one study, it was estimated that people with a personality disorder were seven times more likely to have an anxiety disorder or mood disorder than were those with no personality disorder and four times as likely to have a substance use disorder (Lenzenweger, Lane, Loranger & Kessler, 2007). The links of personality disorder with anxiety, mood and substance are particularly pronounced for the cluster B personality disorders. As a result, personality disorders are commonly encountered in treatment settings, with as many as 40 percent of outpatients meeting the diagnostic criteria for a personality disorder (Newton-Howes et al., 2010). See figure 12.1 for rates of specific personality disorders in the general community as compared to treatment settings.

**TABLE 12.1** Key features of the DSM-5 personality disorders

	Key features	Included in the alternative DSM-5 model for personality disorders?
<i>Cluster A (odd/eccentric)</i>		
Paranoid	Distrust and suspiciousness of others	No
Schizoid	Detachment from social relationships and restricted range of emotional expression	No
Schizotypal	Lack of capacity for close relationships, magical thinking or odd beliefs or speech, and eccentric behaviour	Yes
<i>Cluster B (dramatic/erratic)</i>		
Antisocial	Disregard for and violation of the rights of others	Yes
Borderline	Instability of interpersonal relationships, self-image and affect, as well as marked impulsivity	Yes
Histrionic	Excessive emotionality and attention seeking	No
Narcissistic	Grandiosity, need for admiration and lack of empathy	Yes
<i>Cluster C (anxious/fearful)</i>		
Avoidant	Social inhibition, feelings of inadequacy and hypersensitivity to negative evaluation	Yes
Dependent	Excessive need to be taken care of, submissive behaviour and fears of separation	No
Obsessive-compulsive	Preoccupation with order, perfection and control	Yes

**FIGURE 12.1** Rates of DSM personality disorders in the community and in treatment settings

**Source:** Prevalence estimates for community settings are drawn from Trull, Jahng, Tomko, Wood, & Sher (2010); Samuels et al. (2002). Prevalence estimates for treatment settings are drawn from Zimmerman, Rothschild and Chelminski (2005).

When comorbid personality disorders are present, they are associated with more severe symptoms, poorer social functioning and worse treatment outcome for many psychological disorders. For example, findings of a meta-analysis of 24 studies indicate that a comorbid personality disorder doubles the risk of poor outcomes in depressive disorders (Newton-Howes, Tyrer, & Johnson, 2006). Comorbid personality disorders also predict worse outcomes for anxiety disorders (Ansell et al., 2011).

## Assessment of DSM-5 personality disorders

DSM-5 provides a list of criteria for each of the personality disorders, and structured interviews have been developed to cover these criteria. Table 12.2 shows interrater reliabilities of the personality disorders, as assessed using a structured diagnostic interview administered by experts (Zanarini et al., 2000). Although diagnoses of most personality disorders have adequate or good reliability when structured interviews are used, schizoid personality disorder is still characterised by relatively low interrater reliability.

**TABLE 12.2** Interrater reliability for personality disorders as assessed by structured interview

Diagnosis	Interrater reliability (correlation)
Paranoid	.86
Schizoid	.69
Schizotypal	.91
Antisocial	.97
Borderline	.90
Histrionic	.83
Narcissistic	.88
Avoidant	.79
Dependent	.87
Obsessive-compulsive	.85

*Source:* Structured interview estimates from Zanarini et al. (2000).

Of concern, most clinicians do not use structured interviews to assess personality. To examine the reliability of diagnoses for the three most common personality disorders when assessed with an unstructured clinical interview, the DSM-5 field trials involved clinicians from seven different major psychiatric centres. Clinicians were encouraged to use their typical clinical interviews, but two simple techniques were used to enhance those interviews. First, all patients completed a self-rating of their symptoms that was shared with the clinicians before the interview. Second, after the clinical interview, the interviewers completed a computerised checklist of symptoms to help organise their diagnostic decisions. Although the interrater reliability for borderline personality disorder was adequate, the reliability estimates of anti-social and obsessive-compulsive personality disorders were not adequate — clinicians who had relied on an unstructured interview frequently disagreed about whether the personality disorder was present (Regier, Kuhl, & Kupfer, 2013).

A recent review study was conducted examining population prevalence of personality disorder and associations with health care utilisation in Australia, England, Scotland, Western Europe, Wales, Norway and the United States (Quirk et al., 2016). The prevalence of any personality disorders was common (4.4 percent–21.5 percent) among all these countries. Overall, a positive relationship was found between cluster A and cluster B personality disorders with physical health comorbidities, in particular cardiovascular diseases and arthritis. Additionally, this review also reported higher levels of usage of health care resources among people with personality disorders.

Another study in Australia examined the risks of having a specific personality disorder (PD) and associations with one or more physical conditions as well as having other mental illnesses (see Jackson & Burgess, 2004). Those with probable personality disorder, almost twice more likely, reported experiencing one or more physical health conditions (such as asthma, chronic bronchitis, anaemia, high blood pressure, heart trouble or kidney disease). This study also showed that people with some particular personality disorders, such as borderline personality disorder, experience a higher rate of other mental illness, such as mood or anxiety disorders, and experience greater disability. For more details, see Jackson and Burgess (2004).

Beyond the relatively low agreement rates, clinicians using unstructured clinical interviews often miss personality disorder diagnoses (Zimmerman & Mattia, 1999). In one study, researchers conducted structured diagnostic interviews to assess borderline personality disorder with more than 400 patients and then compared the results to the clinical diagnoses that those patients had received. Among patients who met the criteria for borderline personality disorder according to the structured clinical interviews, less than half had received that clinical diagnosis from their treatment provider (Zimmerman & Mattia, 1999).

Despite this strong evidence that structured diagnostic interviews can enhance diagnostic accuracy and reliability, many clinicians still prefer to use their own unstructured assessments rather than a structured interview. They often argue that a well-trained clinician might have a refined notion of personality disorders and that we should rely on their judgements before those based on a structured interview. Research does not support this idea. Diagnoses based on structured interviews do a better job of predicting functioning and symptoms five years later than do those based on unstructured clinical interviews (Samuel et al., 2013).

Beyond decisions about whether to use a structured diagnostic interview, diagnosticians need to decide whether to interview someone who knows the patient. The diagnostic criteria specify that people with personality disorders tend to see themselves in distorted ways (Thomas, Turkheimer, & Oltmanns, 2003) and so it should not be surprising that clients' reports of their personality disorder symptoms tend to differ from the reports of their friends and families (Klonsky, Oltmanns, & Turkheimer, 2002). Interviews with people who know the patient well improve the accuracy of diagnosis (Bernstein et al., 1997) and enhance the ability to predict social outcomes across a several-year follow-up (Klein, 2003). However, fewer than 10 percent of published studies of personality disorders gather data from people other than the person being diagnosed (Bornstein, 2003).

## Problems with the DSM-5 approach to personality disorders

There are some major concerns about the DSM-5 approach to personality disorders. For example, a growing body of research suggests that these disorders are not as stable as the definition implies and there are extremely high rates of comorbidity among the personality disorders.

### Personality disorders are not stable over time

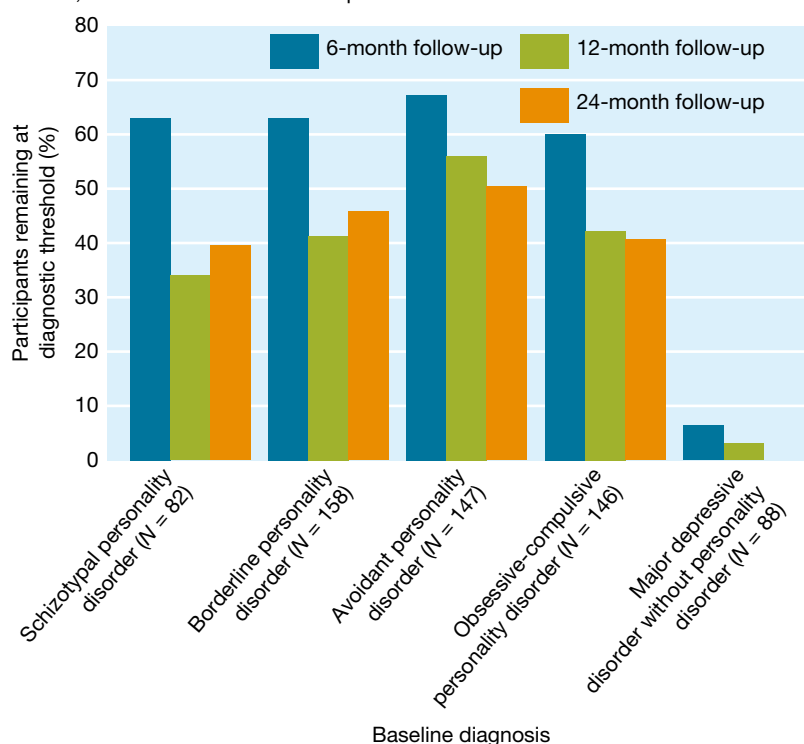
Although the very definition of personality disorders suggests that they should be stable over time, figure 12.2 shows that about half of the people diagnosed with a personality disorder at one point in time had achieved remission (i.e., did not meet the criteria for the same diagnosis) when they were interviewed two years later (McGlashan et al., 2005). When patients diagnosed with personality disorder were followed for 16 years, 99 percent of personality disorder diagnoses remitted (Zanarini, Frankenburg, Reich, & Fitzmaurice, 2011). Personality disorder symptoms appear to be most common during adolescence and then decline into the 20s (Johnson et al., 2000), with even more declines by late life (Balsis, Gleason, Woods, & Oltmanns, 2007). These results, then, indicate that many of the personality disorders may not be as enduring as the DSM asserts.

Even though these symptoms are not as persistent as the definition of personality disorder implies, these diagnoses do seem important for predicting long-term outcomes. First, many people still have some symptoms after remission, just not at the levels required for diagnosis. Second, even after remission, many problems with functioning persist. Baseline diagnoses of personality disorders predict lower functioning and more depression even 10 to 15 years later (Hong et al., 2005; Morey et al., 2012). Third, even years after remission, the risk of relapse remains high — symptoms of personality disorders often

wax and wane over time (Zanarini et al., 2011). Hence, even after remission, a diagnosis of personality disorder can predict ongoing difficulty in achieving a truly satisfying lifestyle.

**FIGURE 12.2**

Test-retest stability for personality disorders and major depressive disorder across 6-, 12- and 24-month follow-up interviews



*Source:* Grilo et al. (2004); Shea et al. (2002).

### Personality disorders are highly comorbid

A second major problem in classifying personality disorders arises from their comorbidity with each other. The opening scenario of Maria illustrates this issue: Maria met the diagnostic criteria not only for borderline personality disorder but also for paranoid personality disorder. More than 50 percent of people diagnosed with a personality disorder meet the diagnostic criteria for another personality disorder (Lenzenweger et al., 2007). Some of the personality disorders involve similar types of concerns. For example, as we will see, the diagnostic criteria for schizotypal, avoidant and paranoid personality disorders all emphasise difficulty in forming close relationships and so it is not surprising that these diagnoses often co-occur. The high rates of overlap among the personality disorders is discouraging when we try to interpret the results of research that compares people who have a specific personality disorder with some control group. If, for example, we find that people with borderline personality disorder differ from healthy people, is our finding related to borderline personality disorder or to personality disorders in general?

In sum, the DSM system might not be ideal for classifying personality disorders, because of the lack of test-retest stability and the high rates of comorbidity. These types of concerns led the DSM-5 Committee on Personality and Personality Disorders to recommend a fundamentally revised approach to personality disorders. We turn to this system next.

### Alternative DSM-5 model for personality disorders

To address issues with the traditional approach to diagnosing personality disorders, the DSM-5 Committee on Personality and Personality Disorders suggested a bold overhaul. They recommended reducing the

number of personality disorders, incorporating personality trait dimensions and diagnosing personality disorders on the basis of extreme scores on personality trait dimensions. The American Psychiatric Association (APA) Board of Trustees decided to retain the personality disorder system that was in place in DSM-IV-TR but included the alternative approach in the appendix of the DSM-5 manual. Although the approach to personality disorders that appears in the main text dominates clinical practice, it is worth considering the merits of the alternative approach.

As shown in table 12.1, the alternative DSM-5 Model for Personality Disorders includes only 6 of the 10 DSM-5 personality disorders. Schizoid, histrionic and dependent personality disorders are excluded from the alternative system because they rarely occur. Paranoid personality disorder, which frequently overlapped with other personality disorders in its occurrence, is also excluded from the alternative system. As shown in table 12.2, interrater reliability is adequate for each of the six personality disorders included in the alternative system when assessed with a structured diagnostic interview.

In the alternative DSM-5 model, diagnoses are based on personality traits. The system includes two types of dimensional scores: 5 **personality trait domains** and 25 more specific **personality trait facets**, as shown in table 12.3. Each dimension can be rated using self-report items (Krueger, Derringer, Markon, Watson, & Skodol, 2012). For example, the Anxiousness facet is assessed with items like ‘I worry a lot about terrible things that might happen’. These personality trait domains and facets are closely related to a very influential model of personality called the five-factor model (McCrae & Costa, 1990).

**TABLE 12.3** The 5 personality trait domains and 25 facets in the DSM-5

Facet	I. Negative affectivity (vs. emotional stability)
1. Anxiousness	
2. Emotional lability	
3. Hostility	
4. Perseveration	
5. Separation insecurity	
6. Submissiveness	
	II. Detachment (vs. extraversion)
7. Anhedonia	
8. Depressivity	
9. Intimacy avoidance	
10. Suspiciousness	
11. Withdrawal	
12. Restricted affectivity	
	III. Antagonism (vs. agreeableness)
13. Attention seeking	
14. Callousness	
15. Deceitfulness	
16. Grandiosity	
17. Manipulativeness	
	IV. Disinhibition (vs. conscientiousness)
18. Distractibility	
19. Impulsivity	
20. Irresponsibility	
21. (Lack of) rigid perfectionism	
22. Risk taking	

- 23. Eccentricity
- 24. Cognitive perceptual dysregulation
- 25. Unusual beliefs and experiences

Extreme scores on these dimensions have been shown to distinguish people with personality disorders from those in the general population (Clark & Livesley, 2002; Samuel & Widiger, 2008). In the alternative DSM-5 model, personality disorders are to be diagnosed when a person shows persistent and pervasive impairments in self and interpersonal aspects of functioning from early adulthood. If a personality disorder is to be diagnosed, the clinician uses the person's profile of personality domain and facet scores to decide which personality disorder might best fit. For example, obsessive-compulsive personality disorder is defined by high scores on rigid perfectionism, along with high scores on at least two of the three dimensions of perseveration, intimacy avoidance and restricted affectivity.

The focus on personality traits has several advantages. Some of the key strengths include the following.

- Those who meet the criteria for a given personality disorder can vary a good deal from one another in their personality traits and the severity of their symptoms. By using the personality trait dimension system, clinicians can specify which personality traits are of most concern for a given client. The 25 dimensional scores provide richer detail than do the personality disorder diagnoses.
- Personality trait ratings tend to be more stable over time than are personality disorder diagnoses (McGlashan, et al., 2005).
- Personality trait dimensions are related to many aspects of psychological adjustment and even physical outcomes. For example, many psychological disorders such as anxiety, depression and somatic symptom disorder can be related to elevations of personality traits such as negative affectivity (Kotov, Gamez, Schmidt, & Watson, 2010). Personality dimensions also robustly predict important interpersonal outcomes such as the quality of friendships and partnerships, performance in one's career, health behaviours and happiness, and they even predict life expectancy (Hopwood & Zanarini, 2010; Ozer & Benet-Martinez, 2006; Roberts, Kuncel, Shiner, Caspi, & Goldberg, 2007). A system based on personality traits helps link the DSM with a broad research literature on personality (Tyrer, 2013).

Despite the strengths of the alternative DSM-5 model for personality disorders, we focus on the approach to personality disorders that appears in the main body of the DSM-5. Debate about the best classification systems should not lead us to underestimate the importance of identifying personality disorders. Personality disorders are prevalent and they cause severe impairments.

## 12.2 Common risk factors across the personality disorders

**LEARNING OUTCOME 12.2** Describe commonalities in the risk factors across the personality disorders.

Theorists over the past 100 years have tried to understand why the chronic, wide-ranging symptoms of personality disorders develop. Although psychoanalytic and behavioural theory placed emphasis on parenting and early developmental influences, more recent work provides evidence for a strong biological component to these syndromes as well. To provide some background on how nature and nurture could be related to the personality disorders, we will focus on two major studies. Both studies examined all 10 DSM-5 personality disorders. Although much of the research on personality disorders has focused on patient samples (and so might be most relevant for those with severe personality disorders), both of these studies relied on large community samples.

Let's begin with the Children in the Community Study, designed to assess the links between childhood adversity and personality disorders. In this study, researchers recruited a representative sample of

639 families with children between ages 1 and 11. Families and children were first interviewed in 1975, again in 1983–1985, for a third time between 1991 and 1993, and then the offspring were interviewed when they reached age 33. Records from child protective services documented childhood maltreatment for 31 of the children and another 50 cases of abuse were identified through self-report. In the assessments conducted in the 1970s and 1980s, researchers conducted interviews with the parents and children to assess two aspects of parenting style: aversive parental behaviour (e.g., harsh punishment, loud arguments) and lack of parental affection (e.g., little time together, poor supervision, poor communication). At the third and fourth interviews, the researchers conducted structured diagnostic interviews to assess personality disorders and the young adults were interviewed about experiences of childhood neglect. Offspring who experienced childhood abuse or neglect were compared with a control group matched on age, parental education and parental psychiatric disorders (Johnson, Cohen, Brown, Smailes, & Bernstein, 1999).

Findings of this study suggested that personality disorders were strongly related to early adversity. Childhood abuse or neglect was related to significantly higher risk of six of the personality disorders: paranoid, antisocial, borderline, narcissistic, dependent and obsessive-compulsive personality disorders. Some of the personality disorders were particularly related to childhood abuse or neglect. For example, children who experienced abuse or neglect were 18 times as likely to develop narcissistic personality disorder and more than six times as likely to develop paranoid, borderline or dependent personality disorder, as were those with no history of abuse or neglect. Parenting style also predicted six of the DSM-5 personality disorders after controlling for childhood behavioural problems and parental psychopathology. Offspring who had experienced aversive or unaffectionate parental styles were several times more likely to develop a personality disorder than were those who had not experienced those parental styles (Johnson, Cohen, Chen, Kasen, & Brook, 2006). Clearly, many people with personality disorders have had difficult experiences during their childhood.

Let's now consider a study designed to estimate the heritability of personality disorders. Researchers used the Norwegian birth registry to recruit a representative sample of twins. To improve diagnostic reliability, the researchers combined self-ratings with interview-based ratings of personality disorder severity. As shown in table 12.4, heritability estimates for all of the personality disorders were at least moderately high. These statistics indicate that we need to consider the biological as well as the social roots of personality disorders.

**TABLE 12.4** Estimated heritability of the personality disorders

Disorder	Estimated heritability
Paranoid	.66
Schizoid	.55
Schizotypal	.72
Antisocial	.69
Borderline	.67
Histrionic	.63
Narcissistic	.71
Avoidant	.64
Dependent	.66
Obsessive-compulsive	.77

**Source:** Kendler, Myers, Torgersen, Neale, & Reichborn-Kjennerud (2007); Gjerde et al. (2012); Torgersen et al. (2012); Torgersen et al. (2000).

The magnitude of the genetic influence on personality disorders suggests that we should be cautious throughout this chapter as we think about parenting and early environment — many parents of those with personality disorders are likely to experience at least mild personality problems themselves. We will discuss the idea that correlation may not mean causation later in the chapter, as we consider studies that have tried to fit together the genetic and environmental contributions to personality disorders.

We now turn to the clinical description of the personality disorders and to the risk factors that shape their development more specifically than the general influences described here. As we do so, our coverage of aetiology will depend on the depth of research available. There is a vast literature on borderline personality disorder and antisocial personality disorder, but for other disorders, little is known beyond the genetic vulnerability and early adversity factors described here. Because so little is known about the aetiology of paranoid, schizoid or histrionic personality disorders, we will not discuss the aetiology of these three disorders further.

## 12.3 Clinical description and aetiology of the odd/eccentric cluster

**LEARNING OUTCOME 12.3** Discuss the clinical description and aetiology of the odd/eccentric cluster of DSM-5 personality disorders.

The odd/eccentric cluster of personality disorders includes paranoid personality disorder, schizoid personality disorder and schizotypal personality disorder. The symptoms of these three disorders bear some similarity to the types of bizarre thinking and experiences seen in schizophrenia. In the cluster A personality disorders, though, the bizarre thinking and experiences are less severe than they are in schizophrenia.

### CLINICAL CASE

#### Danielle

Danielle and her partner sought marital therapy because their day-to-day life was filled with acrimonious quarrels. During the course of the interview, it became clear that Danielle was often angry and hostile towards her partner over small daily incidents. For instance, she thought that he chewed loudly as a way of upsetting her and that he rolled over in bed to deliberately wake her up at night. At a deeper level, they had experienced several periods in which she became deeply fearful that he was having an affair and despite his protestations to the contrary, her fears were very difficult to calm.

In an individual interview, it became clear that she rarely trusts anyone. Her husband was only one of many sources of concern and annoyance for Danielle. She was in a constant state of vigilance thinking about people's motivations and how they might hurt her. Danielle worried that the neighbours played their stereo loudly just to get at her and that her boss assigned her challenging jobs because he wanted to see her squirm. She had long since stopped using banks as she felt that the management might scheme to keep her money and she refused to use email because she was concerned that the government would monitor her computer transactions. Although bright and witty, she was unable to identify women whom she could trust, leaving her friendless. Indeed, every relationship in her life seemed to be coloured by the sense that the other person could harm her or take her resources. Although Danielle felt extremely stressed by her social troubles, she did not believe that she had contributed to any of the conflicts and she felt highly victimised by these difficult interpersonal circumstances.

#### QUESTIONS

1. What are the possible complications in the treatment of Danielle?
2. As a psychologist, how would you engage Danielle in the treatment?

## Paranoid personality disorder

People with **paranoid personality disorder** (see the clinical case of Danielle) are suspicious of others, including strangers, casual acquaintances and even family members. They expect to be mistreated or exploited, and thus are secretive and continually on the lookout for signs of trickery and abuse. They are often hostile and angry in response to perceived insults. Co-workers tend to see them as difficult and critical. Their lives tend to be filled with conflict and those conflicts can be quite long-lasting. Sadly, the conflicts tend to perpetuate their paranoia — their frequent battles provide evidence that people just cannot be trusted.

### DSM-5

#### DSM-5 criteria for paranoid personality disorder

People with paranoid personality disorder experience four or more of the following signs of distrust and suspiciousness from early adulthood across many contexts:

- unjustified suspiciousness of being harmed, deceived or exploited
- unwarranted doubts about the loyalty or trustworthiness of friends or associates
- reluctance to confide in others because of suspiciousness
- the tendency to read hidden meanings into the benign actions of others
- bears grudges for perceived wrongs
- angry reactions to perceived attacks on character or reputation
- unwarranted suspiciousness of the partner's fidelity.

This disorder is different from paranoid schizophrenia because other symptoms of schizophrenia, such as hallucinations, are not present and there is less impairment in social and occupational functioning. Also absent is the cognitive disorganisation that is characteristic of schizophrenia. It differs from delusional disorder because full-blown delusions are not present. Paranoid personality disorder co-occurs most often with schizotypal, borderline and avoidant personality disorders.

## Schizoid personality disorder

Like Joe, described in the clinical case, people with **schizoid personality disorder** do not desire or enjoy social relationships and they usually have no close friends. They appear dull, bland and aloof and have no warm, tender feelings for other people. They rarely experience strong emotions, are not interested in sex and have few pleasurable activities. Indifferent to praise or criticism, people with this disorder are loners who pursue solitary interests.

### DSM-5

#### DSM-5 criteria for schizoid personality disorder

People with schizoid personality disorder experience four or more of the following signs of aloofness and flat affect from early adulthood across many contexts:

- lack of desire for or enjoyment of close relationships
- almost always prefers solitude to companionship
- little interest in sex
- few or no pleasurable activities
- lack of friends
- indifference to praise or criticism
- flat affect, emotional detachment or coldness.

## CLINICAL CASE

### Joe

Joe, a 53-year-old unmarried former farmer, was referred for therapy by his general practitioner due to concerns about Joe's disconnection from most aspects of the social world. Joe reluctantly agreed to see a therapist. He had not worked in several years and survived on a small disability pension. He said that he found it unpleasant to shop for groceries because he didn't like having other people around him. The landlady at his boarding house persisted in trying to introduce him to women, even though he had declared his lack of interest in each and every one of the 10 women she had pestered him to meet. He stated that he did not like talking and in therapy sessions, he was often silent for long periods. He did talk about his sense that he had no real interpersonal ties and that he experienced little emotion other than mild irritability. Indeed, he was unable to describe a single person or activity that made him happy. After six weeks of therapy, Joe announced that he didn't think that he was the type of person who would feel any better from talking about things and that he had decided to spend his remaining savings to purchase a small cabin in a remote area in Brisbane. He seemed content with his decision, stating that by living there, he could successfully escape from almost all social interaction. He moved the next week.

### QUESTIONS

1. Do you think Joe is at higher risk of developing other psychological disorders?
2. What are the difficulties in the treatment of Joe?

## Schizotypal personality disorder

The defining features of **schizotypal personality disorder** include eccentric thoughts and behaviour, interpersonal detachment and suspiciousness. People with this disorder might have odd beliefs or magical thinking — for instance, the belief that they can read other people's minds and see into the future. It is also common for them to have ideas of reference (the belief that events have a particular and unusual meaning for them personally). For example, they might feel that a TV program conveys a special message designed for them. They are often suspicious of others and concerned that others might hurt them. They might also have recurrent illusions (inaccurate sensory perceptions), such as sensing the presence of a force or a person that is not actually there. In their speech, they might use words in an unusual and unclear fashion — for example, they might say 'not a very talkable person' to mean a person who is not easy to talk to. Their behaviour and appearance might also be eccentric — for example, they might talk to themselves or wear dirty and dishevelled clothing. Their affect appears constricted and flat, and they tend to be aloof from others. A study of the relative importance of these symptoms for diagnosis found that suspiciousness, ideas of reference and illusions were most telling (Widiger, Frances, & Trull, 1987). Although most do not develop delusions (convictions in patently absurd beliefs) or schizophrenia, some people diagnosed with schizotypal personality disorder develop more severe psychotic symptoms over time, and a small proportion do develop schizophrenia over time (Raine, 2006). People with schizotypal personality disorder may experience brief episodes of delusions or hallucinations, but it is not as frequent or intense as what is experienced in schizophrenia. Also, it is possible to make someone with schizotypal personality disorder aware of the difference between their distorted ideas and reality. This would be difficult with someone who is diagnosed with schizophrenia and is experiencing severe episodes of delusions or hallucinations, as they are not in touch with reality.

### DSM-5 criteria for schizotypal personality disorder

People with schizotypal personality disorder have five or more of the following signs of unusual thinking, eccentric behaviour and interpersonal deficits from early adulthood across many contexts:

- ideas of reference
- odd beliefs or magical thinking, for example, belief in extrasensory perception
- unusual perceptions
- odd thought and speech
- suspiciousness or paranoia
- inappropriate or restricted affect
- odd or eccentric behaviour or appearance
- lack of close friends
- social anxiety and interpersonal fears that do not diminish with familiarity.

A fair amount of research has focused on the causes of the odd thinking, bizarre behaviour and interpersonal difficulties that appear in schizotypal personality disorder. As noted above, genetic factors and childhood adversity are likely both involved. Beyond this, the biological vulnerability for schizotypal personality disorder appears to overlap with the genetic vulnerability for schizophrenia (Siever & Davis, 2004). That is, family studies and adoption studies have shown that the relatives of people with schizophrenia are at increased risk for schizotypal personality disorder (Nigg & Goldsmith, 1994; Tienari et al., 2003). Studies also have consistently shown that people with schizotypal personality disorder have deficits in cognitive and neuropsychological functioning that are similar to but milder than those seen in schizophrenia (McClure, Barch, Flory, Harvey, & Siever, 2008; Raine, 2006). Furthermore, and again paralleling findings from schizophrenia research, people with schizotypal personality disorder have enlarged ventricles and less temporal lobe grey matter (Dickey, McCarley, & Shenton, 2002).

## 12.4 Clinical description and aetiology of the dramatic/erratic cluster

**LEARNING OUTCOME 12.4** Discuss the clinical description and aetiology of the dramatic/erratic cluster of DSM-5 personality disorders.

The disorders in the dramatic/erratic cluster — antisocial personality disorder, borderline personality disorder, histrionic personality disorder and narcissistic personality disorder — are characterised by symptoms that range from highly inconsistent behaviour to inflated self-esteem, rule-breaking behaviour and exaggerated emotional displays, including anger outbursts. More is known about the aetiology of personality disorders in the dramatic/erratic cluster than those in the other clusters.

Informally, the lay public often uses the terms *antisocial personality disorder* and *psychopathy* interchangeably. Antisocial behaviour, such as law breaking, is a core component of both, but the two syndromes differ in important ways. One difference is that antisocial personality disorder is included in the DSM, whereas psychopathy is not.

### Antisocial personality disorder: clinical description

The core feature of **antisocial personality disorder (APD)** is a pervasive pattern of disregard for the rights of others. The person with APD is distinguished by aggressive, impulsive and callous traits. DSM-5 criteria specify the presence of conduct disorder: people with APD often report a history of such symptoms as truancy, running away from home, frequent lying, theft, arson and deliberate destruction of property by early adolescence. As adults, people with APD show irresponsible behaviour such as working inconsistently, breaking laws, being irritable and physically aggressive, defaulting on debts,

being reckless and impulsive, and neglecting to plan ahead. They show little regard for truth and little remorse for their misdeeds, even when those actions hurt family and friends.

#### DSM-5

##### DSM-5 criteria for antisocial personality disorder

- People with antisocial personality disorder are aged at least 18.
- People with antisocial personality disorder display evidence of conduct disorder before age 15.
- People with antisocial personality disorder display a pervasive pattern of disregard for the rights of others from the age of 15 as shown by at least three of the following:
  1. repeated law breaking
  2. deceitfulness, lying
  3. impulsivity
  4. irritability and aggressiveness
  5. reckless disregard for own safety and that of others
  6. irresponsibility as seen in unreliable employment or financial history
  7. lack of remorse.

Men are about five times more likely than are women to meet criteria for APD (Oltmanns & Powers, 2012). About three-quarters of people with APD meet the diagnostic criteria for another disorder, with substance abuse being very common (Lenzenweger et al., 2007). Not surprisingly, then, high rates of APD are observed in drug and alcohol rehabilitation facilities (Sutker & Adams, 2001). About three-quarters of convicted felons meet the diagnostic criteria for APD.

#### RESEARCH EXAMPLE

##### Co-occurring disorders in forensic mental health

A recent study in Australia investigated the prevalence of co-occurring disorders, such as antisocial personality disorder, psychotic disorder, mood disorder, anxiety disorder, substance use disorder (current and lifetime) and offending, among 130 male offenders in forensic mental health service (Ogloff, Talevski, Lemphers, Wood, & Simmons, 2015). The result showed that 43.1 percent of this population met the criteria for antisocial personality disorder. In addition, 33.1 percent of them met the criteria for at least one current substance use disorder; while, 77.7 percent met the criteria for at least one lifetime substance use disorder. As such, the prevalence of a current or lifetime psychotic disorder, mood disorder or anxiety disorder among this population was high (26.0 to 45.4). The majority of this population were experiencing co-occurring mental disorders and a significant minority met the criteria for antisocial personality disorder. Compared to those with mental illness alone, the participants who had co-occurring mental illness and substance use disorders, and additionally a diagnosis of antisocial personality disorder, committed more serious and frequent offending. An important implication of this study was that the substance disorders must be addressed by professionals working with this population to ensure recovery from the mental illness and to diminish the probability of offending. Considering this complexity, to address the needs of this population and to have a successful treatment, an integration of more effective treatments is critical.

##### QUESTION

What challenges might a practitioner need to consider when integrating treatments?

## Psychopathy: clinical description

The concept of **psychopathy** predates the DSM diagnosis of antisocial personality disorder. In his book *The Mask of Sanity*, Hervey Cleckley (1976) drew on his clinical experience to formulate diagnostic

criteria for psychopathy. The criteria for psychopathy focus on the person's thoughts and feelings. One of the key characteristics of psychopathy is poverty of emotions, both positive and negative: psychopathic people have no sense of shame and their seemingly positive feelings for others are merely an act. They are superficially charming and use that charm to manipulate others for personal gain. Their lack of anxiety might make it impossible for them to learn from their mistakes and their lack of remorse leads them to behave irresponsibly and often cruelly towards others. The rule-breaking behaviour of a person with psychopathy is performed impulsively, as much for thrills as for financial gain. Researchers have argued that three core traits underpin these different symptoms: boldness, meanness and impulsivity (Patrick, Fowles, & Krueger, 2009). The most commonly used scale to assess psychopathy is the Psychopathy Checklist-Revised (PCL-R; Hare, 2003). Ratings on this 20-item scale are made based on an interview and review of criminal records and mental health charts.

#### CLINICAL CASE

##### Fred and Rosemary

One of the worst serial killer couples in history are Fred and Rosemary, who both came from problematic backgrounds. Fred's stepdaughter, Charmaine, and her mother were the first two victims of the couple.

The couple had a perverted marriage — Fred encouraged Rosemary to become a prostitute and even approved of her having sex with her own father. They had a room at their house for Rosemary to entertain clients. The room had a red light in front to warn the children to not interrupt and peep holes through which Fred could watch.

Between 1973 and 1987 the couple raped and killed eight women including their neighbours' children. When they could not find a victim they abused and killed their own daughter. Fred and Rosemary were caught in 1994. That was when police started to search for their daughter who had disappeared in 1987. Fred was found guilty of 21 murders. Rosemary was charged for 10 murders, although she never confessed.

##### QUESTIONS

1. How you can explain this case in terms of psychopathology?
2. By which theory may you be able to better explain their psychopathology (e.g., psychoanalysis, behavioural or even biological)?

There are two chief differences between the criteria for APD and the definition of psychopathy as reflected on the PCL-R. First, even though the PCL-R covers many of the criteria for APD, the scale differs from the DSM-5 criteria for APD in including more affective symptoms, such as shallow affect and lack of empathy (Hare & Neumann, 2006). Second, the DSM-5 criteria for antisocial personality disorder differ from psychopathy criteria in the requirement that a person develop symptoms before age 15. The differences in definition lead to considerable divergence between the two syndromes; many people diagnosed with DSM APD will not obtain high scores on the Psychopathy Checklist (Rutherford, Cacciola, & Alterman, 1999).

#### FOCUS ON DISCOVERY 12.1

##### Media images of psychopathy: will the real psychopath please stand up?

Media images of psychopathy vary considerably, ranging from portrayals of ruthless murderers to charming business tycoons to white-collar criminals. The media is quick to label psychopathy in mass murderers and other ruthless, violent offenders. At the same time, the idea that people with psychopathy use their charm, boldness and lack of empathy to climb their way into the board room, where they are influencing the culture of current business practice, also has become quite widespread in the media. Stories abound of remorseless capitalists who bilk their customers of money through white-collar crime, like Jordan Belfort who was portrayed in the film *The Wolf of Wall Street* (Smith & Lilienfeld, 2013).

Media images abound of remorseless capitalists engaging in white-collar crime, such as Leonardo DiCaprio's portrayal of Jordan Belfort in *The Wolf of Wall Street*.



Data is available regarding each of these stereotypes. Some, but certainly not all, people with high levels of psychopathy engage in violence as a means of achieving their goals (Reidy, Shelley-Tremblay, & Lilienfeld, 2011). Regarding corporate success, a widely cited study did suggest that employees in a large corporate management training program obtained PCL-R scores that were somewhat higher than the general population, but only 3 percent of the employees scored above the PCL-R threshold for psychopathy (Babiak, Neumann, & Hare, 2010). Moreover, psychopathy scores were not elevated in one sample of white-collar criminals (Ragatz, Fremouw, & Baker, 2012). It is not safe to presume that someone is psychopathic just because he or she engages in violent or unethical behaviour or is highly ambitious and successful. Careful diagnosis depends on evaluating whether an entire syndrome is present.

#### QUESTIONS

1. How do you differentiate antisocial personality disorder from psychopathy?
2. What are the more common features between antisocial personality disorder and psychopathy?

### Aetiology of antisocial personality disorder and psychopathy

As we review research on the aetiology of APD and psychopathy, keep in mind two issues that make findings a little hard to integrate. First, research has been conducted on persons diagnosed in different ways — some with APD and some with psychopathy. Second, most research on APD and psychopathy has been conducted on persons who have been convicted as criminals. Thus, the results of this research might not be applicable to those with APD who are not criminals or who avoid arrest. Indeed, among people with high psychopathy levels, cognitive and psychophysiological deficits are more likely among people who have been convicted than those who have not been caught (Ishikawa, Raine, Lencz, Bihrie, & Lacasse, 2001).

More than any other area of personality disorder research, the work on APD often conjointly considers biology with social and psychological risk factors. In this section, we will see this in two ways. First, in considering the social correlates of APD, we will describe how genes and social risk factors work together. Second, as we discuss psychological models of APD, we will note several studies that have used brain imaging to test these models. To capture these integrated models, we will deviate from our organisational approach in other sections of the book, where we tend to separate neurobiological and psychological models.

### **Interactions of genes and the social environment**

Major studies support the role of the social environment as a key factor in APD. Parenting qualities of negativity, inconsistency and low warmth predict antisocial behaviour (Marshall & Cooke, 1999; Reiss et al., 1995). Substantial prospective research also shows that broader social factors, including poverty and exposure to violence, predict antisocial behaviour (Loeber & Hay, 1997). For example, among adolescents with conduct disorder, those who are impoverished are twice as likely to develop APD as are those from higher socioeconomic status backgrounds (Lahey, Loeber, Burke, & Applegate, 2005). There is little question that childhood adversity can set the stage for the development of APD.

The effects of early adversity might be particularly negative for those who are genetically vulnerable to APD. Across multiple studies, a polymorphism of the *MAO-A* gene has been found to predict psychopathy among males who had experienced childhood physical or sexual abuse or maternal rejection (Byrd & Manuck, 2014; Caspi et al., 2002). The effects of growing up in a difficult environment on APD may be amplified by genetic factors.

Adoption research has also shown that genetic, behavioural and family influences are very hard to disentangle (Ge et al., 1996). That is, the genetically influenced antisocial behaviour of the child can provoke harsh discipline and lack of warmth, even in adoptive parents, and these parental characteristics in turn exacerbate the child's antisocial tendencies. Nonetheless, the findings of many studies indicate that social influences such as harsh discipline and poverty robustly predict APD even after controlling for genetic risk (Jaffee, Strait, & Odgers, 2012).

### **Psychological risk: insensitivity to threat and to others' emotions**

People with psychopathy seem unable to learn from experience; they often repeat misconduct that has been harshly punished, even if it resulted in jail time. They seem immune to the anxiety that keeps most of us from breaking the law, lying or injuring others. Cleckley argued that people with psychopathy do not learn to avoid trouble because they are insensitive to threats.

A large body of work relates psychopathy to deficits in the experience of fear and threat. At rest, people with psychopathy have lower-than-normal levels of skin conductance, and their skin conductance is less reactive when they are confronted with or anticipate an aversive stimulus (Lorber, 2004). This low skin conductance reactivity to aversive stimuli (loud tones) at age 3 was found to predict psychopathy scores at age 28 (Glenn, Raine, Venables, & Mednick, 2007).

The behavioural model draws from this idea to suggest that the rule breaking observed in psychopathy stems from deficits in developing conditioned fear responses. In an interesting test of this theory, researchers used brain activity as a way to examine what happens with classical conditioning in which an unconditioned stimulus (painful pressure) was repeatedly paired with neutral pictures (the conditioned stimuli). To measure responses to the conditioned stimulus after these repeated pairings, the researchers measured the activity of the amygdala and other brain regions involved in emotion (Birbaumer et al., 2005). The amygdala is a brain region that is strongly implicated in emotion reactivity (see figure 4.4) and activity of this region has been found to be heightened in several disorders that involve intense emotionality, including mood disorders and anxiety disorders. After conditioning, healthy control participants showed increases in amygdala activity when viewing the neutral pictures.

People with high psychopathy scores, though, did not show this expected increase in amygdala activity. These findings suggest that people with psychopathy show weakened classical conditioning to aversive stimuli.

Beyond their general lack of response to threat, the person with psychopathy might become even more unresponsive to threats when trying to gain a reward, such as money or other resources. In a study demonstrating this phenomenon, participants played a computerised card game in which they earned 5 cents for each face card that appeared; if a nonface card appeared, the participant lost 5 cents (Newman, Patterson, & Kosson, 1987). Participants could quit the game anytime they wanted. The game was rigged so that over time, the probability of losing increased. People with high psychopathy scores continued to play the game much longer than did people with low psychopathy, even when they were being punished. Findings like these suggest that psychopathy is related to inattentiveness to threats when pursuing a goal (Zeier & Newman, 2013). Consistent with these findings, antisocial behaviour is associated with deficits in regions of the prefrontal cortex that are involved in attending to negative information during goal pursuit (Ermer, Cope, Nyalakanti, Calhoun, & Kiehl, 2012; Yang & Raine, 2009).

In contrast to a general insensitivity to threat, some researchers believe that a lack of empathy, defined as the capacity to share the emotional reactions of others, could be the central deficit driving the callous exploitation of others observed in psychopathy (Blair, 2005). Several types of research provide support for this theory. When asked to identify the emotion conveyed in videos of strangers, men with psychopathy do very poorly in recognising others' fear, even though they recognise other emotions well (Brook & Kosson, 2013). To test whether their lack of empathy extends to insensitivity towards others' victimisation, researchers have studied responses to pictures of victimisation events (e.g., a break-in, a physical attack). People with high psychopathy levels show less psychophysiological or neural response to viewing pictures of victimisation than do those with low psychopathy levels (Harenski, Harenski, Shane, & Kiehl, 2010; Levenston, Patrick, Bradley, & Lang, 2000).

## Borderline personality disorder

Borderline personality disorder (BPD) has been a major focus of interest for several reasons: it is very common in clinical settings, very hard to treat and associated with recurrent periods of suicidality. The core features of **borderline personality disorder** are impulsivity and instability in relationships and mood. For example, people with this disorder may shift from blissful happiness to outraged explosions in the blink of an eye. Attitudes and feelings towards other people can also change drastically, inexplicably and very quickly, particularly from passionate idealisation to contemptuous anger (Trull et al., 2008). As in the case of Maria, which opened this chapter, the intense anger of people with BPD often damages relationships. People with BPD are overly sensitive to small signs of emotions in others (Lynch et al., 2006). Their unpredictable, impulsive and potentially self-damaging behaviour might include gambling, reckless spending, indiscriminate sexual activity and substance abuse. People with BPD often have not developed a clear and coherent sense of self — they sometimes experience major swings in such basic aspects of identity as their values, loyalties and career choices. They cannot bear to be alone, have fears of abandonment and experience chronic feelings of depression and emptiness. They may experience transient psychotic and dissociative symptoms when stressed.

Suicidal behaviour is all too common in BPD. Many people with this disorder make multiple suicide attempts during their lifetime (Boisseau, Yen, Markowitz et al., 2013). In one 20-year follow-up study, about 7.5 percent of people with BPD died from suicide (Linehan & Heard, 1999). People with BPD are also particularly likely to engage in non-suicidal self-injury (see focus on discovery 3.6). For example, they might slice their legs with a razor blade or burn their arms with cigarettes — behaviours that are harmful but unlikely to cause death. At least two-thirds of people with BPD will engage in self-mutilation at some point during their lives (Stone, 1993).

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After he was diagnosed with borderline personality disorder in 2010, Brandon Marshall, a highly successful American football player, created the Brandon Marshall Foundation to help improve awareness of mental health, to reduce stigma and to provide funding for treatment.



People with BPD are very likely to have comorbid post-traumatic stress disorder and mood disorders, substance-related disorders and eating disorders (McGlashan et al., 2000). When present, comorbid conditions predict greater likelihood that BPD symptoms will be sustained over several years (Zanarini, Frankenburg, Hennen, Reich, & Silk, 2004).

A colourful account by Jonathan Kellerman, a clinical psychologist and successful mystery writer, gives a good sense of what people with BPD are like:

They're the chronically depressed, the determinedly addictive, the compulsively divorced, living from one emotional disaster to the next. Bed hoppers, stomach pumpers, freeway jumpers and sad-eyed bench-sitters with arms stitched up like footballs and psychic wounds that can never be sutured. Their egos are as fragile as spun sugar, their psyches irretrievably fragmented, like a jigsaw puzzle with crucial pieces missing. They play roles with alacrity, excel at being anyone but themselves, crave intimacy but repel it when they find it. Some of them gravitate toward stage or screen; others do their acting in more subtle ways ... Borderlines go from therapist to therapist, hoping to find a magic bullet for the crushing feelings of emptiness. They turn to chemical bullets, gobble tranquilizers and antidepressants, alcohol and cocaine. (Kellerman, 1989, pp. 113–114)

Fortunately, research on new treatments for BPD, discussed later in the chapter, supports a more positive outlook than Kellerman offers.

### DSM-5 criteria for borderline personality disorder

People with borderline personality disorder display five or more of the following signs of instability in relationships, self-image and impulsivity from early adulthood across many contexts:

- frantic efforts to avoid abandonment
- unstable interpersonal relationships in which others are either idealised or devalued
- unstable sense of self
- self-damaging, impulsive behaviours in at least two areas, such as spending, sex, substance abuse, reckless driving and binge eating
- recurrent suicidal behaviour, gestures or self-injurious behaviour (e.g., cutting self)
- marked mood reactivity
- chronic feelings of emptiness
- recurrent bouts of intense or poorly controlled anger
- during stress, a tendency to experience transient paranoid thoughts and dissociative symptoms.

### Aetiology of borderline personality disorder

Many different risk factors may contribute to the development of BPD. We discuss neurobiological factors, research that conjointly considers genetic vulnerability and social factors, and Linehan's diathesis–stress theory, which integrates neurobiological and social factors.

#### Neurobiological factors

Some neurobiological factors are thought to increase risk for the full syndrome of BPD, whereas others are thought to help explain only the intense emotionality or impulsivity of those with the disorder (Siever, 2000). Relevant to a general dysregulation, people with BPD demonstrate lower serotonin function than do controls (Soloff, Meltzer, Greer, Constantine, & Kelly, 2000). Relevant to the emotion dysregulation, people with BPD show increased activation of the amygdala to emotional pictures (Hazlett et al., 2012; Silbersweig et al., 2007), in contrast to the diminished amygdala activity shown among fearless psychopaths. Deficits in the prefrontal cortex are thought to contribute to impulsivity and people with BPD show deficits in the prefrontal cortex (Minzenberg, Fan, New, Tang, & Siever, 2008; van Elst, 2003) and disrupted connectivity between the prefrontal cortex and the amygdala (New et al., 2007). Taken together, findings suggest that multiple facets of neurobiology may converge to create the complex array of symptoms that comprise BPD.

#### Social factors: childhood abuse in the context of genetic vulnerability

In our earlier discussion of risk factors common across personality disorders, we noted that BPD has been tied to extremely high rates of childhood abuse or neglect, as well as to high estimates of heritability. How can we fit these two risk factors together? To consider this question, researchers studied 197 monozygotic (MZ) twin pairs in which one twin, but not the other, reported childhood abuse (Bornovalova et al., 2013). If abuse, rather than genetic vulnerability, is driving BPD, twins who experienced abuse should have a higher rate of BPD than their co-twins who were not abused. This was not the case. The twin pairs had similar levels of BPD. That is, childhood abuse did not predict BPD after genetic risk was controlled. In a second twin study, childhood traumatic experiences accounted for less than 1 percent in the variance among those who developed BPD after accounting for family characteristics (Berenz et al., 2013).

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People with borderline personality disorder often engage in self-injurious behaviour.



Even though a history of abuse is common for those with BPD, these findings indicate that abuse may not be the driving force that sets this personality disorder in motion. How can we make sense of the high rates of abuse among those with BPD, then? Researchers are still working on this puzzle, but it is important to consider the idea that abuse often happens in a context of many different problems. Genetically driven impulsivity, emotionality or risk-seeking in the parents could increase the risk that both abuse and BPD will occur. Findings highlight the complexity of abuse, as it often occurs within a matrix of risk factors. Although much remains unknown, it is worth noting that common sense still holds — child abuse has many deleterious effects. These have been shown in other studies and even in other studies of twins discordant for abuse (Nelson et al., 2002).

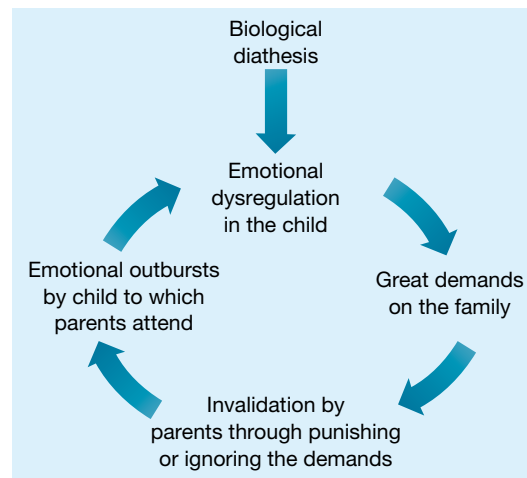
#### **Linehan's diathesis–stress theory**

Marsha Linehan proposes that BPD develops when people who have difficulty controlling their emotions because of a biological diathesis (possibly genetic) are raised in a family environment that is invalidating. That is, a diathesis of emotional dysregulation interacts with experiences of invalidation to promote the development of BPD. In an invalidating environment, the person's feelings are discounted and disrespected — that is, the person's efforts to communicate feelings are disregarded or even punished.

The two main hypothesised factors — emotional dysregulation and invalidation — interact with each other in a dynamic fashion (see figure 12.3). For example, the emotionally dysregulated child makes enormous demands on his or her family. The exasperated parents ignore or even punish the child's outbursts, which leads the child to suppress his or her emotions. The suppressed emotions build up to an explosion, which then gets the attention of the parents. Thus, the parents end up reinforcing the very behaviours that they find aversive. Many other patterns are possible, of course, but what they have in common is a vicious circle, a constant back-and-forth between dysregulation and invalidation.

**FIGURE 12.3**

Marsha Linehan's diathesis–stress theory of borderline personality disorder



## Histrionic personality disorder

The key feature of **histrionic personality disorder** is overly dramatic and attention-seeking behaviour. People with this disorder often use their physical appearance, such as unusual clothes, make-up or hair colour, to draw attention to themselves. Despite their expressions of extravagant and intense emotions, they are thought to be emotionally shallow. For example, someone with this disorder might gush about and call a person his or her best friend, only to have trouble remembering a conversation with that person the next day. They are self-centred, overly concerned with their physical attractiveness and uncomfortable when not the centre of attention. They can be inappropriately sexually provocative and seductive, and are easily influenced by others. Their speech is often impressionistic and lacking in detail. For example, they might state a strong opinion, yet be completely unable to support it. (Patient: 'She was absolutely the greatest.' Interviewer: 'What did you like best about her?' Patient: 'Gosh, I'm not sure I could describe that.')

### DSM-5

#### DSM-5 criteria for histrionic personality disorder

People with histrionic personality disorder display five or more of the following signs of excessive emotionality and attention seeking from early adulthood across many contexts:

- strong need to be the centre of attention
- inappropriate sexually seductive behaviour
- rapidly shifting and shallow expression of emotions
- use of physical appearance to draw attention to self
- speech that is excessively impressionistic and lacking in detail
- exaggerated, theatrical emotional expression
- overly suggestible
- misreads relationships as more intimate than they are.

## Narcissistic personality disorder

People with **narcissistic personality disorder** have a grandiose view of their qualities and are preoccupied with fantasies of great success (as demonstrated by Dev in the clinical case below). They are more

than a little self-centred — they require almost constant attention. Their interpersonal relationships are disturbed by their lack of empathy, by their arrogance coupled with feelings of envy, by their habit of taking advantage of others and by their feelings of entitlement and expectations that others will do special favours for them. Partnerships are often challenged by their excessive need to be in charge. They tend to seek out high-status partners whom they idealise and proudly show off, only to change partners if given an opportunity to be with a person of higher status. Fame and wealth are often overly valued as a way to gain the admiration of others. With their focus on gaining admiration, appearance matters — in one study, independent observers were able to make snap judgements of narcissism from photographs with some accuracy, most typically by noticing the expensive clothes and overinvestment in appearance of those with narcissistic traits (Vazire, Naumann, Rentfrow, & Gosling, 2008). At the same time that they exude overconfidence, an underlying vulnerability is apparent in their envy and need to be admired (Miller, Hoffman, Campbell, & Pilkonis, 2008). Just as they will bask in compliments and praise, they also tend to be overly reactive to criticism. They are highly likely to be vindictive and aggressive when faced with a competitive threat or a put-down (Bushman & Thomaes, 2011).

Frank Lloyd Wright, one of the most influential American architects, displayed at least some narcissistic traits. He is quoted as saying 'Early in life, I had to choose between honest arrogance and hypocritical humility. I chose honest arrogance and have seen no reason to change'.



### CLINICAL CASE

#### Dev

Dev, a 50-year-old college professor, sought treatment only after urging from his wife. During the interview, Dev's wife noted that he seemed so focused on himself and his own advancement that he often belittled others. Dev was dismissive of these concerns, stating that he had never been the sort of person to tolerate idiots and he could see no reason why he should begin offering such tolerance now — in rapid fire, he described his supervisor, his students, his parents and a set of former friends as lacking the intelligence to merit his friendship. He willingly acknowledged working long hours but stated that his research had the potential to change life for people and that other activities could not be allowed to interfere with his success. The therapist's gentle questioning of whether his expressions of superiority might provoke some interpersonal tension was met with a scathing rebuke.

#### QUESTIONS

1. What are some of Dev's biggest fears?
2. How would Dev's characteristics affect his job performance and decision making?

### DSM-5

#### DSM-5 criteria for narcissistic personality disorder

Presence of five or more of the following signs of grandiosity, need for admiration and lack of empathy from early adulthood across many contexts:

- grandiose view of one's importance
- preoccupation with one's success, brilliance, beauty
- belief that one is special and can be understood only by other high-status people

- extreme need for admiration
- strong sense of entitlement
- tendency to exploit others
- lack of empathy
- envious of others
- arrogant behaviour or attitudes.

Given the associated tendencies towards confidence and ambition, could a small amount of narcissism be adaptive? In one study, 121 scholars with expertise in American leaders rated the relative (subclinical) narcissism of the US presidents. Presidents who were rated as relatively more narcissistic were more likely to be seen as persuasive, able to win the popular vote and to initiate legislation. On the other hand, they were also likely to get into trouble for unethical behaviour (Watts et al., 2013). Those with clinical diagnoses of narcissism would be expected, however, to have more troubles than those with subtle elevations of narcissistic traits.

### **Aetiology of narcissistic personality disorder**

In this section, we begin by discussing a theory of how parenting could contribute to narcissistic personality traits.

#### **Parenting**

Millon (1986) hypothesised that parents who are overly indulgent foster children's belief that they are special (more special than other children) and that behavioural expressions of their specialness will be tolerated by others. Overindulgence of parents can lead to classic or grandiose type narcissism. A more pathological type of narcissism is fragile or vulnerable narcissism. People with fragile narcissism have been psychologically abused and/or experienced neglect in childhood. Grandiose behaviour in fragile narcissism actually serves a defensive function against underlying feelings of inferiority, inadequacy and emptiness. The grandiose sense of self-importance is an overcompensation against feelings of inferiority and smallness. This is one reason that people with fragile narcissism are often anxious; they are fearful of rejection and when they are abandoned they fall into depression.

#### **Self-psychology**

In his two books, *The Analysis of the Self* (1971) and *The Restoration of the Self* (1977), Heinz Kohut developed a model of narcissism based on self-psychology, a variant of psychodynamic theory. He started from the clinical observation that the person with narcissistic personality disorder projects self-importance, self-absorption and fantasies of limitless success on the surface as a mechanism to survive their painful feelings. Kohut theorised that these characteristics in fact mask a very fragile self-esteem. People with narcissistic personality disorder strive to bolster their sense of self-worth through unending quests for respect from others. Inflated self-worth and denigration of others, then, are seen as defences against feelings of shame. Research studies do support the hypothesis that people diagnosed with narcissistic personality disorder experience shame more frequently than do those without personality disorder (Ritter et al., 2014).

#### **Social-cognitive model**

A model of narcissistic personality disorder developed by Carolyn Morf and Frederick Rhodewalt (2001) is built around two basic ideas: (1) people with this disorder have fragile self-esteem, in part because they are trying to maintain the belief that they are special and (2) interpersonal interactions are important to them for bolstering self-esteem rather than for gaining closeness or warmth. In other words, they are captive to the goal of maintaining a grand vision of themselves and this goal pervades their experiences. Like the self-psychology model, this model focuses on the fragile self-esteem of the narcissist but places greater emphasis on social and cognitive mechanisms driving this syndrome.

To assess the idea that people with narcissistic personality disorder are trying to maintain grandiose beliefs about themselves, Morf and Rhodewalt have examined biases in how people with this disorder

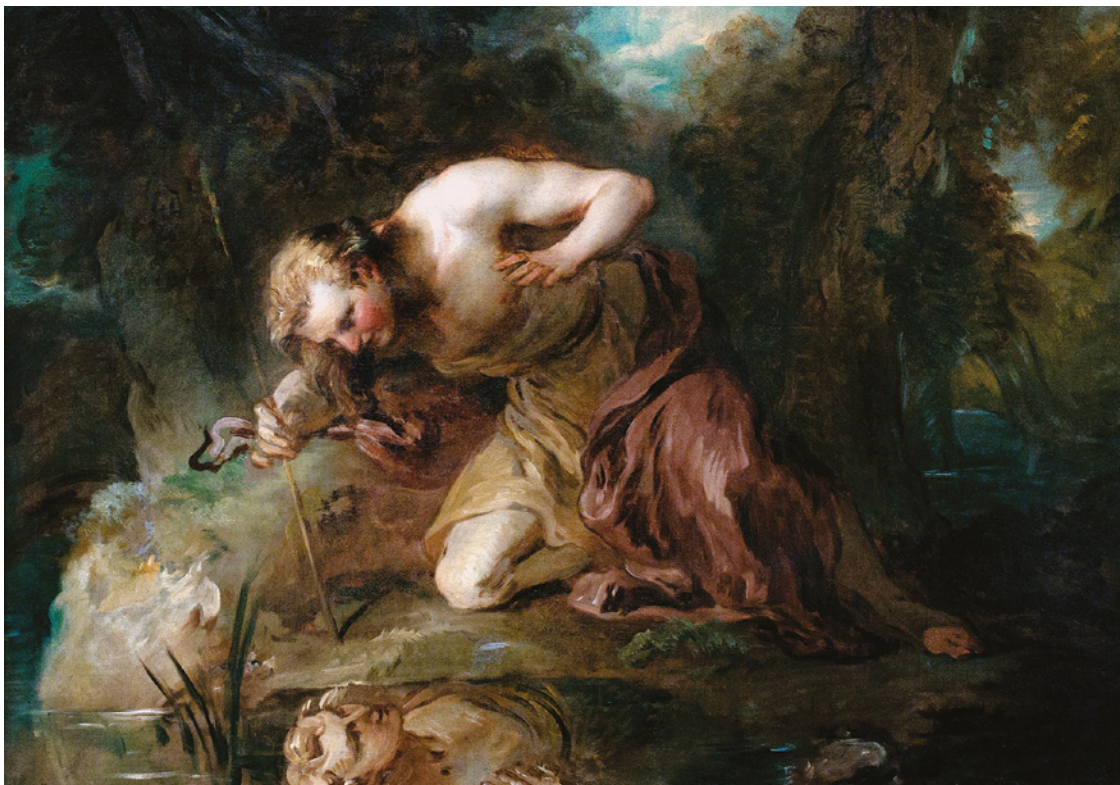
rate themselves in various settings. For example, in laboratory studies, people with narcissistic personality disorder overestimate their attractiveness to others and their contributions to group activities. ('Others must be jealous of me; I've been responsible for the lion's share of our progress here today.') In some studies, researchers have provided people with feedback that they were successful on a task (regardless of their actual performance) and then asked participants to rate the reasons why they were successful. In these types of studies, people with narcissistic personality disorder attribute successes to their abilities rather than to chance or luck to a greater extent than do those without a personality disorder. So a set of studies suggest that people with narcissistic personality disorder show cognitive biases that would help maintain grandiose beliefs about the self.

To assess whether people with narcissistic personality disorder have fragile self-esteem, Morf and Rhodewalt (2001) have reviewed studies of how much self-esteem depends on external feedback. For example, when falsely told they have done poorly on an IQ test, they show much more reactivity than others do; similarly, they show more reactivity to being told they have succeeded at something.

According to this theory, when people with narcissistic personality disorder interact with others, their primary goal is to bolster their own self-esteem. This goal influences how they act towards others in several ways. First, they tend to brag a lot; this often works well initially, but over time, repeated bragging comes to be perceived negatively by others (Paulhus, 1998). Second, when someone else performs better than they do on a task that is relevant to self-esteem, they will denigrate the other person, even to that person's face. This framework makes it easy to understand why people with narcissistic personality disorder engage in behaviours that alienate others; their sense of self depends on being admired, not in gaining or maintaining closeness (Campbell, Bosson, Goheen, Lakey, & Kernis, 2007).

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Narcissistic personality disorder draws its name from the Greek mythological figure Narcissus, who fell in love with his own reflection, was consumed by his own desire and was transformed into a flower. Narcissus by Francois Le Moyne (1688–1733).



## 12.5 Clinical description and aetiology of the anxious/fearful cluster

**LEARNING OUTCOME 12.5** Discuss the clinical description and aetiology of the anxious/fearful cluster of DSM-5 personality disorders.

The anxious/fearful cluster includes avoidant personality disorder, dependent personality disorder and obsessive-compulsive personality disorder. People with these disorders are prone to worry and distress.

### Avoidant personality disorder

People with **avoidant personality disorder** are so fearful of criticism, rejection and disapproval that they will avoid jobs or relationships to protect themselves from negative feedback. In social situations, they are restrained and timid because of an extreme fear of saying something foolish, being embarrassed, blushing or showing other signs of anxiety. They believe they are incompetent and inferior to others, and they are reluctant to take risks or try new activities. Even though they would like to form close relationships, their fears often make it difficult for them to do so.

People with avoidant personality disorder often avoid interpersonal interactions, as they find them to be so stressful.



#### DSM-5

##### DSM-5 criteria for avoidant personality disorder

People with avoidant personality disorder display a pervasive pattern of social inhibition, feelings of inadequacy and hypersensitivity to criticism as shown by four or more of the following from early adulthood across many contexts:

- avoidance of occupational activities that involve significant interpersonal contact, because of fears of criticism or disapproval
- unwilling to get involved with people unless certain of being liked
- restrained in intimate relationships because of the fear of being shamed or ridiculed

- preoccupation with being criticised or rejected
- inhibited in new interpersonal situations because of feelings of inadequacy
- views self as socially inept, unappealing or inferior
- unusually reluctant to try new activities because they may prove embarrassing.

Avoidant personality disorder often co-occurs with social anxiety disorder (see chapter 4), probably because the diagnostic criteria for these two disorders are so similar (Skodol et al., 1995). The genetic vulnerability for avoidant personality disorder and social anxiety disorder appears to overlap (Reichborn-Kjennerud, Czajkowski, Torgersen et al., 2007). Some have argued that avoidant personality disorder might actually be a more chronic variant of social anxiety disorder (Alden, Laposa, Taylor, & Ryder, 2002). Aetiological variables, then, might overlap with those of social anxiety, discussed in chapter 4.

As is true for social anxiety disorders, several other types of disorders are often comorbid with avoidant personality disorder. About 80 percent of people with avoidant personality disorder, like Leon in the following clinical case, have comorbid major depression. Alcohol abuse is also common among people with this disorder (McGlashan et al., 2000).

#### CLINICAL CASE

##### Leon

Leon, a 45-year-old man, sought treatment for depression. During the interview, Leon described feeling depressed and uncomfortable socially for as long as he could remember. At age five, he would experience intense anxiety when he was with other children and his mind would 'go blank' if he had to speak in front of others. He grew up dreading birthday parties, teachers' classroom questions and meeting new children. Although he was able to play with some of the children in his neighbourhood, he had never asked a woman out on a date or developed a 'best friend'. He took a job at the post office after graduation because it involved little social interaction. (Adapted from Spitzer, Gibbon, Skodol, Williams, & First, 1994)

#### QUESTIONS

1. How is Leon's personality different from someone with schizoid personality disorder?
2. In which area — interpersonal or social/occupational functioning — would Leon experience most difficulties?

## Dependent personality disorder

The core feature of **dependent personality disorder** is an excessive reliance on others. People with dependent personality disorder have an intense need to be taken care of, which often leads them to feel uncomfortable with being alone. They subordinate their own needs to ensure that they do not threaten the protective relationships they have established. When a close relationship ends, they urgently seek another relationship to replace it. They see themselves as weak and they turn to others for support and decision making. The clinical case of Matthew provides an example of dependent personality disorder. People with this disorder particularly fear being alone.

#### CLINICAL CASE

##### Matthew

Matthew, a 34-year-old man, sought treatment after breaking up with a girlfriend. His mother, with whom he lived, had disapproved of his marriage plans because his girlfriend came from a different religious background. Matthew felt that he could not marry his girlfriend without his mother's approval. Although

he cancelled the engagement, he was angry with his mother and feared that she would never approve of anyone he wanted to marry. He said that he feared disagreeing with his mother because he did not want to have to fend for himself. (Adapted from Spitzer, 2002)

### QUESTIONS

1. If fear of loss of support or disapproval is one of Matthew's fears, how would it affect the treatment process?
2. What would a psychologist need to do to address this fear and encourage Matthew to express his disagreement?

## DSM-5

### DSM-5 criteria for dependent personality disorder

People with dependent personality disorder have an excessive need to be taken care of, as shown by the presence of at least five of the following from early adulthood across many contexts:

- difficulty making decisions without excessive advice and reassurance from others
- need for others to take responsibility for most major areas of life
- difficulty disagreeing with others for fear of losing their support
- difficulty doing things on own or starting projects because of lack of self-confidence
- doing unpleasant things as a way to obtain the approval and support of others
- feelings of helplessness when alone because of fears of being unable to care for self
- urgently seeking new relationship when one ends
- preoccupation with fears of having to take care of self.

The DSM diagnostic criteria portray people with dependent personality disorder as being very passive (e.g., having difficulty initiating projects or doing things on their own, allowing others to make decisions for them). Research indicates, however, that people with dependent personality disorder actually can do what is necessary to maintain a close relationship; this might involve being very deferential and passive, but it might involve taking active steps to preserve the relationship (Bornstein, 1997). Also contrary to the idea that this disorder is characterised by passivity, men with higher levels of dependency, perhaps because of their insecurity when their partnerships are threatened, are at elevated risk of perpetrating domestic violence (Bornstein, 2006).

People with dependent personality disorder are likely to develop depression after interpersonal losses (Hammen et al., 1995) and when they are depressed, they show more suicidality than do other depressed patients (Bolton, Belik, Enns, Cox, & Sareen, 2008). They are also at elevated risk for developing anxiety disorders and bulimia (Bornstein, 2012).

In addition to the genetic and early adversity findings described above, theorists argue that overprotective parents may reinforce children for dependency, while authoritarian discipline may limit the opportunities for children to develop feelings of self-efficacy (Bornstein, 1992). Findings of several studies support the idea that dependent personality traits are related to overprotective and authoritarian parenting (Bornstein, 1992).

## Obsessive-compulsive personality disorder

The person with **obsessive-compulsive personality disorder** is a perfectionist, preoccupied with details, rules and schedules. Although order and perfectionism have their adaptive sides, particularly in fostering success in complex occupational goals, people with this disorder often pay so much attention to detail that they fail to finish projects (as illustrated in the clinical case of Faiza). They are more oriented towards work than pleasure and social relationships often suffer as the pursuit of perfection in the

workplace takes time away from family and friends. They have inordinate difficulty making decisions (lest they err) and allocating time (lest they focus on the wrong thing). Their interpersonal relationships are often troubled because they demand that everything be done the right way — their way. Generally, they are serious, rigid, formal and inflexible, especially regarding moral issues. They are unable to discard worn-out and useless objects, even those with no sentimental value, and they are likely to be excessively frugal to a level that causes concern among those around them.

Obsessive-compulsive personality disorder is distinct from obsessive-compulsive disorder, despite the similarity in names. The personality disorder does not include the obsessions and compulsions that define the latter. Nonetheless, the two conditions often co-occur (Skodol et al., 1995) and seem to have some overlapping genetic vulnerability (Taylor, Asmundson, & Jang, 2011).

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For the person with obsessive-compulsive personality disorder, the overly perfectionistic quest for order may interfere with being productive.



#### DSM-5

##### **DSM-5 criteria for obsessive-compulsive personality disorder**

People with obsessive-compulsive personality disorder have an intense need for order, perfection and control, as shown by the presence of at least four of the following from early adulthood across many contexts:

- preoccupation with rules, details and organisation to the extent that the point of an activity is lost
- extreme perfectionism interferes with task completion
- excessive devotion to work to the exclusion of leisure and friendships
- inflexibility about morals and values
- difficulty discarding worthless items
- reluctance to delegate unless others conform to one's standards
- miserliness
- rigidity and stubbornness.

## CLINICAL CASE

### Faiza

Faiza, a 22-year-old woman, was ecstatic to attain a research assistant position with a highly accomplished scientist just after graduation. She was planning to pursue a career in science and the position fit perfectly with her long-held interests. Despite Faiza's initial enthusiasm, things soon soured with her new boss. When asked to collect data using methods that she already knew well, she began by creating large spreadsheets of the planned process and then shifted gears and invested in elaborate software for project management. No matter how hard she tried, she could not move from project planning into actual data collection. When faced with the idea of writing up a project description for the university ethics board review, she became buried as she wrote over 50 pages of detailed notes on potentially relevant issues but then could not find a way to describe the study in the one page allowed on the form. When asked to manage eight undergraduate volunteers who worked with the team, she found it difficult to delegate any tasks to them, instead redoing any of the work that they finished to quell her anxiety about what might happen if they were to make a mistake. Even though she worked 15-hour days, her boss asked her to resign at the end of the 3-month probation period because she had accomplished less than her predecessor, who had worked only 20 hours a week. She was deeply disillusioned by the experience and she sought therapy with a sense that this was just one in a series of events in which she had gotten herself into trouble by losing sight of the forest for the trees.

### QUESTIONS

1. How likely would Faiza have been to come for treatment if her contract was still renewed in her current job?
2. How efficient do you think Faiza feels with her job?

## 12.6 Treatment of personality disorders

**LEARNING OUTCOME 12.6** Describe the available psychological treatments of the DSM-5 personality disorders.

People with a personality disorder do not have particularly strong insight towards their problems; so many of them enter treatment for a condition other than their personality disorder. For example, a person with antisocial personality disorder might seek treatment for substance abuse; a person with avoidant personality disorder might seek treatment for social anxiety disorder; a person with narcissistic personality disorder might seek treatment for depression after being rejected; and a patient with obsessive-compulsive personality disorder might seek help for depression. Clinicians are encouraged to consider whether personality disorders are present because their presence predicts slower improvement in psychotherapy (Crits-Christoph & Barber, 2002; Hollon & DeRubeis, 2003).

Here we consider treatments to address the symptoms of personality disorders. We begin by describing approaches that are relevant across the various personality disorders and then discuss treatments designed for specific personality disorders.

### General approaches to the treatment of personality disorders

Psychotherapy is considered the treatment of choice for personality disorders. In one meta-analysis, psychotherapy was shown to provide small but positive effects compared to treatment as usual across 22 studies of borderline personality disorder and eight of other personality disorders (Budge et al., 2013). Psychotherapy is often supplemented with medications. For example, antidepressants are used to quell some of the depressive or impulsive symptoms that accompany personality disorders (Tyrer & Bateman, 2004).

People with serious symptoms of personality disorders might attend weekly psychotherapy sessions or they might attend a day treatment program that offers psychotherapy, in both group and individual

formats, for several hours per day. Many day treatment programs also provide occupational therapy. The length of day treatment programs varies, but some last several months.

Psychodynamic theory suggests that childhood problems are at the root of personality disorders and so the aim of psychodynamic therapy is to help the patient reconsider those early experiences, and become more aware of how those experiences drive their current behaviours and responses to those early events. For example, a psychodynamic therapist might help a man with obsessive-compulsive personality disorder to the realisation that his need to be perfect is based on painful emotions or unprocessed feelings from childhood. The therapist helps the client to experience the anxiety as well as the feelings underneath the anxiety and to see how they have contributed to development of obsessive-compulsive personality characteristics (Abbass, 2015). Gaskin (2012) presents a systematic review of recent (previous five years) and Australian (previous 10 years) papers on the effectiveness of dynamic psychotherapy in working with clients.

Cognitive theory suggests that negative cognitive beliefs, such as those shown in table 12.5, are at the heart of the personality disorders (Beck & Freeman, 1990). The aim of cognitive therapy, then, is to help a person become more aware of those beliefs and then to challenge maladaptive cognitions. For example, cognitive therapy for a perfectionistic person with obsessive-compulsive personality disorder entails first persuading the patient to accept the essence of the cognitive model — that feelings and behaviours are primarily a function of thoughts. Biases in thinking are then explored, such as when the patient concludes that he or she cannot do anything right because of a small failure in one particular endeavour. The therapist also looks for dysfunctional assumptions or schemas that might underlie the person's thoughts and feelings. Beyond challenging cognitions, Beck's approach to personality disorders incorporates a variety of other cognitive-behavioural techniques.

**TABLE 12.5** Examples of maladaptive cognitions hypothesised to be associated with each personality disorder

Personality disorder	Maladaptive cognitions
Avoidant	If people get to know the real me, they will reject me.
Dependent	I am not capable of making a decision without help and reassurance.
Obsessive-compulsive	I know what's best. If things get disorganised, horrible mistakes will happen.
Paranoid	It is foolish to trust anyone.
Antisocial	People ask for exploitation — they let down their guard.
Narcissistic	I am better than others and people who can't understand that don't deserve my time.
Histrionic	People are there to admire me.
Schizoid	Relationships are more hassle than reward.

*Source:* Adapted from Beck & Freeman (1990).

The traits that characterise the personality disorders are probably too ingrained to change thoroughly. Instead, the therapist — regardless of theoretical orientation — might find it more realistic to change a disorder into a style or a more adaptive way of approaching life (Millon, 1996). Because relatively little is known, we focus on only three personality disorders in the next sections.

## Treatment of schizotypal personality disorder and avoidant personality disorder

Treatments for schizotypal personality disorder draw on the links of this disorder with schizophrenia. More specifically, antipsychotic drugs (e.g., risperidone, trade name Risperdal) have shown effectiveness

with schizotypal personality disorder (Raine, 2006). These medications seem particularly helpful for reducing the unusual thinking. Little research is available on psychological approaches to the treatment of schizotypal personality disorder.

Avoidant personality disorder appears to respond to the same treatments that are effective for those with social anxiety disorder: that is, antidepressant medications as well as cognitive-behavioural treatment (Reich, 2000). Cognitive-behavioural treatment might involve helping a person challenge his or her negative beliefs about social interactions by teaching behavioural strategies for dealing with difficult social situations and by exposure treatment, in which the person gradually takes part in feared social situations. Cognitive-behavioural treatment lasting 20 sessions has been found to be more helpful than psychodynamic treatment for avoidant personality disorder (Emmelkamp et al., 2006). Group versions of cognitive-behavioural treatment have been found to be helpful and may offer chances to practise constructive social interactions in a safe environment (Alden, 1989).

## Treatment of borderline personality disorder

Few clients pose a greater challenge to treat than do those with borderline personality disorder (BPD), regardless of the type of treatment being used. Clients with BPD tend to show their interpersonal problems in the therapeutic relationship as much as they do in other relationships. Because these clients find it inordinately difficult to trust others, therapists find it challenging to develop and maintain the therapeutic relationship. Patients alternately idealise and vilify the therapist, demanding special attention and consideration one moment — such as therapy sessions at odd hours and countless phone calls during crisis periods — and refusing to keep appointments the next; they beg the therapist for understanding and support but insist that certain topics are off-limits.

Suicide is always a serious risk, but it is often difficult for the therapist to judge whether a frantic phone call at 2.00 am from a BPD patient is a call for help or a manipulative gesture designed to test how special the patient is to the therapist and to what lengths the therapist will go to meet the patient's needs at the moment. As in the case of Maria (see the opening scenario at the beginning of this chapter), hospitalisation is often necessary to protect against the threat of suicide. Seeing such clients is so stressful for therapists that many of them regularly consult with another therapist for advice and for support in dealing with their own emotions as they cope with the extraordinary challenges of helping these clients.

We will focus on dialectical behaviour therapy because the benefits of this approach have been demonstrated in more than a dozen research studies. A small set of studies have provided support for psychodynamic therapy (Clarkin, Levy, Lenzenweger, & Kernberg, 2007) and for a longer-term version of cognitive therapy for BPD (Blum et al., 2008; Giesen-Bloo et al., 2006).

**Dialectical behaviour therapy (DBT)** combines client-centred empathy and acceptance with cognitive-behavioural problem solving, emotion-regulation techniques and social skills training (Linehan, 1987). The concept of dialectics comes from German philosopher Georg Wilhelm Friedrich Hegel (1770–1831). It refers to a constant tension between any phenomenon (any idea, event, etc., called the *thesis*) and its opposite (the *antithesis*), which is resolved by creating a new phenomenon (the *synthesis*). In DBT, the term *dialectical* is used in two main ways.

1. In one sense, it refers to the seemingly opposite strategies that the therapist must use when treating people with BPD — accepting them as they are and yet helping them change. (For a closer look at the dialectic between acceptance and change, see focus on discovery 12.2.)
2. In the other sense, it refers to the patient's realisation that splitting the world into good and bad is not necessary; instead, one can achieve a synthesis of these apparent opposites. For example, instead of seeing a friend as either all bad (thesis) or all good (antithesis), the friend can be seen as having both kinds of qualities (synthesis).

Hence, both the therapist and the client in DBT are encouraged to adopt a dialectical view of the world.

### Drawing from personal experience to promote acceptance and change

As described earlier, Marsha Linehan developed dialectical behaviour therapy, the best validated approach for BPD. In a brave move, Linehan decided to speak publicly about her own experiences of BPD (Carey, 2011). Hospitalised at age 17 for her severe suicidality, she found ways to injure herself even when the staff confined her to a seclusion chamber — left alone with no objects, she banged her head against the wall and floor. She remained hospitalised for 26 months. Failed treatments continued for several years, until she found a way out of the struggles on her own — through radical acceptance. She earned a PhD in clinical psychology and she drew on her own personal experiences to help others, in the process becoming one of the most productive researchers in the field of clinical psychology.

Linehan (1987) argues that a therapist treating clients with BPD has to adopt a posture that might seem inconsistent to the Western mind. The therapist must work for change while at the same time accepting the real possibility that no changes are going to occur. Linehan's notions of acceptance are drawn from Zen philosophy and from Rogerian approaches to psychotherapy. Linehan's reasoning is that people with BPD are so sensitive to rejection and criticism that even gentle encouragement to behave or think differently can be misinterpreted as a serious rebuke, leading to extreme emotional reactions. When this happens, the therapist, who may have been revered a moment earlier, is suddenly vilified. Thus, while observing limits — 'I would be very sad if you killed yourself, so I hope very much that you won't' — the therapist must convey to the patient that he or she is fully accepted. This is hard to do if the patient is threatening suicide, showing uncontrolled anger or railing against imagined rebukes from the therapist.

Completely accepting the patient does not mean approving of everything the patient does; rather, it means that the therapist must accept the situation for what it is. And this acceptance, argues Linehan, must be real: the therapist must truly accept clients as they are; acceptance should not be in the service of change, an indirect way of encouraging clients to behave differently. 'Acceptance can transform but if you accept in order to transform, it is not acceptance. It is like loving. Love seeks no reward but when given freely comes back a hundredfold. He who loses his life finds it. He who accepts, changes' (M. Linehan, personal communication, November 16, 1992). Full acceptance does not, in Linehan's view, preclude change. Indeed, she proposes the opposite — that it is the refusal to accept that precludes change.

Linehan's approach also emphasises that clients, too, must accept who they are and what they have been through. Clients are asked to accept that their childhood is now unchangeable, that their behaviours might have caused relationships to end and that they feel emotions more intensely than others do. This approach, it is hoped, will provide a basis for understanding the self and promoting growth.

Marsha Linehan created dialectical behaviour therapy, which combines cognitive-behavioural therapy with acceptance.



#### QUESTIONS

1. Which of the following interventions has been the first to effectively address suicidal thoughts or self-harm behaviour in BPD: mindfulness, emotion regulation, interpersonal effectiveness, distress tolerance strategies or impulse control?
2. What seems to be the most challenging symptom in the treatment of BPD?

The cognitive-behavioural aspect of DBT, conducted both individually and in groups, involves four stages. In the first stage, dangerously impulsive behaviours such as suicidal actions are addressed, with the goal of promoting greater control. The client is taught to identify triggers for these behaviours and to apply coping strategies when the triggers are present. In the second stage, the focus is on learning to modulate the extreme emotionality. This phase might involve coaching to help a person learn to tolerate emotional distress. In this stage, clients are taught to mindfully notice their emotions in a non-judgemental manner, without rushing into impulsive actions. Stage three focuses on improving relationships and self-esteem. Stage four is designed to promote connectedness and happiness. Throughout, clients learn more effective and socially acceptable ways of handling their day-to-day problems. Basically, DBT involves cognitive-behavioural therapy combined with interventions to provide validation and acceptance to the client.

Linehan and colleagues conducted a trial in which clients were randomly assigned either to DBT or to treatment as usual, meaning any therapy available in the community. After 1 year of treatment and again 6 and 12 months later, clients in the two groups were compared on a variety of measures (Linehan, Heard, & Armstrong, 1993). The findings immediately after treatment revealed that DBT was superior to treatment as usual — clients engaged in less intentional self-injurious behaviour, including fewer suicide attempts; dropped out of treatment less; spent fewer days in the hospital; and reported better adjustment and work records. There were, however, no differences in self-reported depression between the two treatment groups, underscoring the extreme difficulty of treating these clients. At the follow-up assessments, the superiority of DBT was sustained. In a meta-analysis of 16 studies conducted since that time, DBT was found to have moderate positive effects in reducing self-injury and suicidality compared to control conditions (Kliem, Kröger, & Kosfelder, 2010).

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## SUMMARY

### **12.1 Explain the DSM-5 approach to classifying personality disorders, key concerns with this approach and the DSM-5 alternative approach to diagnosis.**

Personality disorders are defined by longstanding and pervasive ways of being that cause distress and impairment through their influence on forming and sustaining a positive self-identity and constructive relationships. Many people with personality disorders also meet the diagnostic criteria for comorbid conditions such as depressive, anxiety and substance use disorders as well as other personality disorders. In DSM-5, personality disorders are classified in three clusters: cluster A personality disorders are characterised by odd or eccentric behaviour; cluster B by dramatic, emotional or erratic behaviour; and cluster C by anxious or fearful behaviour. Most personality disorders can be reliably assessed with structured clinical interviews, but unstructured clinical interviews tend to be unreliable and have less predictive validity.

Two concerns have emerged about the DSM-5 personality disorder approach: the personality disorders are not stable over time and they overlap substantially. An alternative DSM-5 model was developed to address these concerns. The alternative model includes only six personality disorders and also includes a dimensional system for evaluating five personality trait domains and 25 more specific personality facets. The alternative model is placed in the appendix of the DSM-5.

### **12.2 Describe commonalities in the risk factors across the personality disorders.**

Genetics and environmental factors have been suggested to contribute to developing personality disorders. All personality disorders are at least moderately heritable. Environmental factors such as childhood abuse or neglect are significantly correlated with 6 of the 10 personality disorders. Individuals who reported higher rates of psychological or emotional abuse, long-term physical abuse or sexual abuse in childhood are more likely to experience personality disorders. Attachment trauma including tragic physical abuse or neglect, very early in life, in particular under age two, adversely affects the brain structure and development and can lead to development of antisocial personality disorder.

In other words, children who are born with a more difficult temperament (i.e., overly sensitive children or children high in neuroticism) are more vulnerable to the development of personality disorders — especially if they grow up in an unstable or chaotic family environment. It is suggested that a warm, supportive, strong relationship in the child's life (even with a teacher, a relative or a friend) can serve as a protective factor against negative environmental adversities. In spite of this fact, these children, and more importantly the ones who have experienced very early attachment trauma or tragedies, need early professional intervention to offset the negative influence of that experience (see the documentary *Child of Rage*).

### **12.3 Discuss the clinical description and aetiology of the odd/eccentric cluster of DSM-5 personality disorders.**

The odd/eccentric cluster of personality disorders (cluster A) includes paranoid personality disorder, schizoid personality disorder and schizotypal personality disorder. People with paranoid personality disorder are suspicious of others, people with schizoid personality disorder are socially aloof, and people with schizotypal personality disorder are eccentric in their thoughts and behaviour. Biological studies indicate that schizotypal personality disorder and schizophrenia are related.

### **12.4 Discuss the clinical description and aetiology of the dramatic/erratic cluster of DSM-5 personality disorders.**

The dramatic/erratic cluster (cluster B) includes antisocial personality disorder (APD), borderline personality disorder (BPD), histrionic personality disorder and narcissistic personality disorder. The key features of APD include violation of rules and a disregard for others' feelings and social norms. Psychopathy is related to antisocial personality disorder but is not defined in the DSM. Psychopathy

criteria focus on internal experiences (such as a poverty of emotion) as well as observable behaviour. BPD is defined by intense emotionality, unstable sense of identity and impulsivity. Histrionic personality disorder is characterised by exaggerated emotional displays. Narcissistic personality disorder is characterised by highly inflated self-esteem but a deep need for admiration.

A harsh family environment and poverty play a role in the development of APD and these social risk factors may be particularly important for those who are at higher genetic risk for the disorder. Psychopathy is related to blunted responses to threat and, as a consequence, to difficulty learning from punishment. This insensitivity to threat may be especially present during goal pursuit. The callous treatment of others might also be linked to a lack of empathy. This lack of anxiety and empathy may drive misconduct without regret.

BPD is related to serotonergic dysfunction and to biological risk factors for emotion dysregulation and impulsivity. Consistent with the greater emotionality, people with BPD demonstrate the increased activity of the amygdala to emotion stimuli. Consistent with the impulsivity, research indicates diminished activity in the prefrontal cortex among people with BPD. People with BPD report elevated rates of abuse, but some of the abuse may be tied to parental genetic vulnerability. Linehan's model integrates the high rates of parental invalidation reported by people with BPD with the biological diathesis for emotional dysregulation.

Across studies, those with narcissistic personality disorder report that their parents were overly indulgent. According to self-psychology theory, those with narcissistic personality disorder inflate their own self-worth to combat feelings of shame. Social-cognitive theory proposes that the behaviour of the person with narcissistic personality disorder is shaped by the goal of maintaining specialness and the belief that the purpose of interpersonal interactions is to bolster self-esteem.

#### **12.5 Discuss the clinical description and aetiology of the anxious/fearful cluster of DSM-5 personality disorders.**

The anxious or fearful cluster of personality disorders (cluster C) includes avoidant personality disorder, dependent personality disorder and obsessive-compulsive personality disorder. People with avoidant personality disorder are timid and often feel inadequate. Those with dependent personality disorder are overly reliant on others, to the extent that they are vulnerable to depression after interpersonal losses. Those with obsessive-compulsive disorder are intensely focused on the details of maintaining order, perfection and control.

Avoidant personality disorder may be a more chronic variant of social anxiety disorder. People with dependent personality disorder often describe their parents as both overprotective and authoritarian. Although the two conditions have distinct symptom profiles, obsessive-compulsive personality disorder and obsessive-compulsive disorder are often comorbid and may have overlapping genetic vulnerability.

#### **12.6 Describe the available psychological treatments of the DSM-5 personality disorders.**

Personality disorders not only adversely affect an individual's functioning at work, in their education or in their relationships, but also significantly affect the people around them. Relationship and interpersonal interaction are severely impacted by personality disorders. Personality disorders can also lead to the development of addictive behaviours, social isolation and even physical health comorbidities. Because of the complexities involved with personality disorders, treatment often lasts longer than a year or even two years. In particular, treatment of borderline personality disorder should be delivered by very experienced and highly trained professionals in the field, since there is the risk of self-harm and suicide.

Cognitive-behavioural therapy targets dysfunctional thoughts, emotion and behaviours in treatment of personality disorders. A multidimensional approach to targeting the complexity of personality disorders can involve other psychotherapy theories, including psychodynamic psychotherapy, schema therapy, dialectic behaviour therapy and interpersonal therapy.

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## KEY TERMS

- antisocial personality disorder (APD)** personality disorder defined by the absence of concern for others' feelings or social norms and a pervasive pattern of rule breaking
- avoidant personality disorder** personality disorder defined by aloofness and extreme sensitivity to potential rejection, despite an intense desire for affiliation and affection
- borderline personality disorder (BPD)** personality disorder defined by impulsiveness and unpredictability, an uncertain self-image, intense and unstable social relationships, and extreme swings of mood
- dependent personality disorder** a personality disorder in which people are overly concerned about maintaining relationships. People with this disorder often allow others to make decisions for them and are reluctant to make demands that could challenge relationships
- dialectical behaviour therapy (DBT)** a therapeutic approach to borderline personality disorder that combines client-centred empathy and acceptance with behavioural problem solving, social skills training and limit setting
- histrionic personality disorder** a personality disorder defined by overly dramatic behaviour, emotional excess and sexually provocative behaviour
- narcissistic personality disorder** personality disorder defined by extreme selfishness and self-centredness; a grandiose view of one's uniqueness, achievements and talents; an insatiable craving for admiration and approval from others; willingness to exploit others to achieve goals; and expectation of much more from others than one is willing to give in return
- obsessive-compulsive personality disorder** personality disorder defined by inordinate difficulty making decisions, hyperconcern with details and efficiency, and poor relations with others due to demands that things be done just so, as well as the person's unduly conventional, serious, formal and stingy emotions
- paranoid personality disorder** a personality disorder defined by expectation of mistreatment at the hands of others, suspicion, secretiveness, jealousy, argumentativeness, unwillingness to accept blame, and cold and unemotional affect
- personality disorder** a group of disorders involving longstanding, inflexible and maladaptive personality traits that impair social and occupational functioning
- personality trait domains** five personality dimensions included in the appendix of DSM-5 to help supplement diagnoses of personality disorders: negative affectivity, detachment, antagonism, disinhibition and psychoticism
- personality trait facets** twenty-five specific personality dimensions included in the appendix of DSM-5 to provide greater detail on the personality trait domains
- psychopathy** a personality syndrome related to antisocial personality disorder but defined by an absence of emotion, impulsivity, manipulateness and irresponsibility
- schizoid personality disorder** a personality disorder defined by emotional aloofness; indifference to the praise, criticism and feelings of others; maintenance of few, if any, close friendships; and solitary interests
- schizotypal personality disorder** personality disorder defined by eccentricity, oddities of thought and perception (magical thinking, illusions, depersonalisation, derealisation), digressive speech involving overelaborations and social isolation; under stress, behaviour may appear psychotic

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## WEBSITES

1. The Millon Personality Group provides information on Millon's fifteen personality styles/ disorders and subtypes. ([www.millonpersonality.com/theory/diagnostic-taxonomy](http://www.millonpersonality.com/theory/diagnostic-taxonomy))
2. Melbourne centre for ISTDP is a Melbourne-based centre for resources on intensive short-term dynamic psychotherapy (ISTDP). (<http://mcistdp.com.au>)

3. California Society for Intensive Short-term Dynamic Psychotherapy is provides information on intensive short-term dynamic psychotherapy. ([www.istdp.com](http://www.istdp.com))

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## REFERENCES

- Abbass, A. (2015). *Reaching through resistance: advanced psychotherapy techniques*. Kansas City: Seven Leaves Press.
- Alden, L. E. (1989). Short-term structured treatment for avoidant personality disorder. *Journal of Consulting and Clinical Psychology*, 57, 756–764.
- Alden, L. E., Laposa, J. M., Taylor, C. T., & Ryder, A. G. (2002). Avoidant personality disorder: Current status and future directions. *Journal of Personality Disorders*, 16, 1–29.
- American Psychiatric Association. (2013). *Diagnostic and Statistical Manual of Mental Disorders 5th edition (DSM-5)* (5th ed.).
- Ansell, E. B., Pinto, A., Edelen, M. O., Markowitz, J. C., Sanislow, C. A., Yen, S., et al. (2011). The association of personality disorders with the prospective 7-year course of anxiety disorders. *Psychological Medicine*, 41, 1019–1028.
- Babiak, P., Neumann, C. S., & Hare, R. D. (2010). Corporate psychopathy: Talking the walk. *Behavioral Sciences and the Law*, 28, 174–193.
- Balsis, S., Gleason, M. E., Woods, C. M., & Oltmanns, T. F. (2007). An item response theory analysis of DSM-IV personality disorder criteria across younger and older age groups. *Psychology and Aging*, 22, 171–185.
- Beck, A. T., & Freeman, A. (1990). *Cognitive therapy for personality disorders*. New York: Guilford Press.
- Berenz, E. C., Amstadter, A. B., Aggen, S. H., Knudsen, G. P., Reichborn-Kjennerud, T., Gardner, C. O., et al. (2013). Childhood trauma and personality disorder criterion counts: A co-twin control analysis. *Journal of Abnormal Psychology*, 122, 1070–1076.
- Bernstein, D. P., Kasapis, C., Bergman, A., Weld, E., Mitropoulou, V., et al. (1997). Assessing Axis II disorders by informant interview. *Journal of Personality Disorders*, 11, 158–167.
- Birbaumer, N., Veit, R., Lotze, M., Erb, M., Hermann, C., Grodd, W., et al. (2005). Deficient fear conditioning in psychopathy: A functional magnetic resonance imaging study. *Archives of General Psychiatry*, 62, 799–805.
- Blair, R. J. R. (2005). Responding to the emotions of others: Dissociating forms of empathy through the study of typical and psychiatric populations. *Consciousness and Cognition*, 14, 698–718.
- Blum, N., John, D. S., Pfohl, B., Stuart, S., McCormick, B., Allen, J. J., et al. (2008). Systems training for emotional predictability and problem solving (STEPPS) for outpatients with borderline personality disorder: A randomized controlled trial and 1-year follow-up. *American Journal of Psychiatry*, 165, 468–478.
- Boisseau, C. L., Yen, S., Markowitz, J. C., Grilo, C. M., Sanislow, C. A., Shea, M. T., et al. (2013). Individuals with single versus multiple suicide attempts over 10 years of prospective follow-up. *Comprehensive Psychiatry*, 54, 238–242.
- Bolton, J. M., Belik, S. L., Enns, M. W., Cox, B. J., & Sareen, J. (2008). Exploring the correlates of suicide attempts among individuals with major depressive disorder: Findings from the National Epidemiologic Survey on Alcohol and Related Conditions. *Journal of Clinical Psychiatry*, 69, 1139–1149.
- Bornovaalova, M. A., Huibregtse, B. M., Hicks, B. M., Keyes, M., McGue, M., & Iacono, W. (2013). Tests of a direct effect of childhood abuse on adult borderline personality disorder traits: A longitudinal discordant twin design. *Journal of Abnormal Psychology*, 122, 180–194.
- Bornstein, R. F. (1992). The dependent personality: Developmental, social, and clinical perspectives. *Psychological Bulletin*, 112, 3–23.
- Bornstein, R. F. (1997). Dependent personality disorder in the DSM-IV and beyond. *Clinical Psychology: Science and Practice*, 4, 175–187.
- Bornstein, R. F. (2003). Behaviorally referenced experimentation and symptom validation: A paradigm for 21st century personality disorder research. *Journal of Personality Disorders*, 17, 1–18.
- Bornstein, R. F. (2006). The complex relationship between dependency and domestic violence: Converging psychological factors and social forces. *American Psychologist*, 61, 595–606.
- Bornstein, R. F. (2012). Dependent personality disorders. In T. A. Widiger (Ed.), *The Oxford Handbook of Personality Disorders*. New York: Oxford University Press.
- Brook, M., & Kosson, D. S. (2013). Impaired cognitive empathy in criminal psychopathy: Evidence from a laboratory measure of empathic accuracy. *Journal of Abnormal Psychology*, 122, 156–166.
- Budge, S. L., Moore, J. T., Del Re, A. C., Wampold, B. E., Baardseth, T. P., & Nienhuis, J. B. (2013). The effectiveness of evidence-based treatments for personality disorders when comparing treatment-as-usual and bona fide treatments. *Clinical Psychology Review*, 33, 1057–1066.
- Bushman, B. J., & Thomaes, S. (2011). When the narcissistic ego deflates, narcissistic aggression inflates. In W. K. Campbell & J. D. Miller (Eds.), *The handbook of narcissism and narcissistic personality disorder: Theoretical approaches, empirical findings, and treatments* (pp. 319–329). Hoboken, NJ: Wiley Press.
- Byrd, A. L., & Manuck, S. B. (2014). MAOA, childhood maltreatment, and antisocial behavior: Meta-analysis of a gene-environment interaction. *Biological Psychiatry*, 75, 9–17.

- Campbell, W. K., Bosson, J. K., Goheen, T. W., Lakey, C. E., & Kernis, M. H. (2007). Do narcissists dislike themselves “deep down inside”? *Psychological Science*, 18, 227–229.
- Carey, B. (2011, June 23). Expert on mental illness reveals her own fight, *New York Times*.
- Caspi, A., McClay, J., Moffitt, T. E., Mill, J., Martin, J., Craig, I. W., Taylor, A., & Poulton, R. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 297, 851–854.
- Clark, L. A., & Livesley, W. J. (2002). Two approaches to identifying the dimensions of personality disorder: Convergence on the five-factor model. In T. A. Widiger & P. T. J. Costa (Eds.), *Personality disorders and the five factor model of personality* (pp. 161–176). Washington, DC: American Psychological Association.
- Clarkin, J. F., Levy, K. N., Lenzenweger, M. F., & Kernberg, O. F. (2007). Evaluating three treatments for borderline personality disorder: A multiwave study. *American Journal of Psychiatry*, 164, 922–928.
- Cleckley, H. (1976). *The mask of sanity* (5th ed.). St. Louis: Mosby.
- Dickey, C. C., McCarley, R. W., & Shenton, M. E. (2002). The brain in schizotypal personality disorder: A review of structural MRI and CT findings. *Harvard Review of Psychiatry*, 10, 1–15.
- Crits-Christoph, P., & Barber, J. P. (2002). Psychological treatments for personality disorders. In P. E. Nathan & J. M. Gorman (Eds.), *A guide to treatments that work*. New York: Oxford University Press.
- Emmelkamp, P. M. G., Benner, A., Kuipers, A., Feiertag, G. A., Koster, H. C., & van Apeldoorn, F. J. (2006). Comparison of brief dynamic and cognitive-behavioural therapies in avoidant personality disorder. *British Journal of Psychiatry*, 189, 60–64.
- Ermer, E., Cope, L. M., Nyalakanti, P. K., Calhoun, V. D., & Kiehl, K. A. (2012). Aberrant paralimbic gray matter in criminal psychopathy. *Journal of Abnormal Psychology*, 121, 649–658.
- Gaskin, C. (2012). *The effectiveness of psychodynamic psychotherapy: A systematic review of recent international and Australian research*. Melbourne: PACFA.
- Ge, X., Conger, R. D., Cadoret, R. J., Neiderhiser, J. M., Yates, W., Troughton, E., et al. (1996). The developmental interface between nature and nurture: A mutual influence model of child antisocial behavior and parent behaviors. *Developmental Psychology*, 32, 574–589.
- Giesen-Bloo, J., van Dyck, R., Spinhoven, P., van Tilburg, W., Dirksen, C., van Asselt, T., et al. (2006). Outpatient psychotherapy for borderline personality disorder: Randomized trial of schema-focused therapy vs transference-focused psychotherapy. *Archives of General Psychiatry*, 63, 649–658.
- Gjerde, L. C., Czajkowski, N., Roysamb, E., Orstavik, R. E., Knudsen, G. P., Ostby, K., et al. (2012). The heritability of avoidant and dependent personality disorder assessed by personal interview and questionnaire. *Acta Psychiatrica Scandinavica*, 126, 448–457.
- Glenn, A. L., Raine, A., Venables, P. H., & Mednick, S. A. (2007). Early temperamental and psychophysiological precursors of adult psychopathic personality. *Journal of Abnormal Psychology*, 116, 508–518.
- Grilo, C. M., Shea, M. T., Sanislow, C. A., Skodol, A. E., Gunderson, J. G., Stout, R. L., et al. (2004). Two-year stability and change of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *Journal of Consulting and Clinical Psychology*, 72, 767–775.
- Hammen, C. L., Burge, D., Daley, S. E., Davila, J., Paley, B., & Rudolph, K. D. (1995). Interpersonal attachment cognitions and prediction of symptomatic responses to interpersonal stress. *Journal of Abnormal Psychology*, 104, 436–443.
- Hare, R. D., & Neumann, C. N. (2006). The PCL-R assessment of psychopathy: Development, structural properties, and new directions. In C. Patrick (Ed.), *Handbook of psychopathy* (pp. 58–88). New York: Guilford Press.
- Hare, R. D. (2003). *The Hare psychopathy checklist* (Rev. ed.). Toronto, Canada: Multi-Health System.
- Harenski, C. L., Harenski, K. A., Shane, M. S., & Kiehl, K. A. (2010). Aberrant neural processing of moral violations in criminal psychopaths. *Journal of Abnormal Psychology*, 119, 863–874.
- Hazlett, E. A., Zhang, J., New, A. S., Zelmanova, Y., Goldstein, K. E., Haznedar, M. M., et al. (2012). Potentiated amygdala response to repeated emotional pictures in borderline personality disorder. *Biological Psychiatry*, 72, 448–456.
- Hollon, S. D., & DeRubeis, R. J. (2003). *Cognitive therapy for depression*. Paper presented at the Annual Conference of the American Psychiatric Association, Philadelphia, PA.
- Hong, J. P., Samuels, J., Bienvenu, O. J., Hsu, F. C., Eaton, W. W., Costa, P. T., Jr., et al. (2005). The longitudinal relationship between personality disorder dimensions and global functioning in a community-residing population. *Psychological Medicine*, 35, 891–895.
- Hopwood, C. J., & Zanarini, M. C. (2010). Borderline personality traits and disorder: Predicting prospective patient functioning. *Journal of Consulting and Clinical Psychology*, 78, 585–589.
- Ishikawa, S. S., Raine, A., Lencz, T., Bihle, S., & Lacasse, L. (2001). Autonomic stress reactivity and executive functions in successful and unsuccessful criminal psychopaths from the community. *Journal of Abnormal Psychology*, 110, 423–432.
- Jackson, H. J., & Burgess, P. M. (2004). Personality disorders in the community: results from the Australian National Survey of Mental Health and Well-being Part III. Relationships between specific type of personality disorder, Axis I mental disorders and physical conditions with disability and health consultations. *Social Psychiatry and Psychiatric Epidemiology*, 39(10), 765–76.
- Jaffee, S. R., Strait, L. B., & Odgers, C. L. (2012). From correlates to causes: Can quasi-experimental studies and statistical innovations bring us closer to identifying the causes of antisocial behavior? *Psychological Bulletin*, 138, 272–295.
- Johnson, J. G., Cohen, P., Brown, J., Smailes, E. M., & Bernstein, D. P. (1999). Childhood maltreatment increases risk for personality disorders during early adulthood. *Archives of General Psychiatry*, 56, 600–606.

- Johnson, J. G., Cohen, P., Chen, H., Kasen, S., & Brook, J. S. (2006). Parenting behaviors associated with risk for offspring personality disorder during adulthood. *Archives of General Psychiatry*, 63, 579–583.
- Johnson, J. G., Cohen, P., Kasen, S., Skodol, A. E., Hamagami, F., & Brook, J. S. (2000). Age-related change in personality disorder trait levels between early adolescence and adulthood: A community-based longitudinal investigation. *Acta Psychiatrica Scandinavica*, 2, 263–275.
- Kellerman, J. (1989). *Silent partner*. New York: Bantam Books.
- Kendler, K. S., Myers, J., Torgersen, S., Neale, M. C., & Reichborn-Kjennerud, T. (2007). The heritability of cluster A personality disorders assessed by both personal interview and questionnaire. *Psychological Medicine*, 37, 655–665.
- Klein, D. N. (2003). Patients' versus informants' reports of personality disorders in predicting 7 1/2-year outcome in outpatients with depressive disorders. *Psychological Assessment*, 15, 216–222.
- Kliem, S., Kröger, C., & Kosfelder, J. (2010). Dialectical behavior therapy for borderline personality disorder: A meta-analysis using mixed-effects modeling. *Journal of Consulting and Clinical Psychology*, 78, 936–951.
- Klonsky, E. D., Oltmanns, T. F., & Turkheimer, E. (2002). Informant-reports of personality disorder: Relations to self-reports and future research directions. *Clinical Psychology: Science and Practice*, 9, 300–311.
- Kohut, H. (1971). *The analysis of the self*. New York: International Universities Press.
- Kohut, H. (1977). *The restoration of the self*. New York: International Universities Press.
- Kotov, R., Gamez, W., Schmidt, F., & Watson, D. (2010). Linking “big” personality traits to anxiety, depressive, and substance use disorders: A meta-analysis. *Psychological Bulletin*, 136(5), 768–821.
- Krueger, R. F., Derringer, J., Markon, K. E., Watson, D., & Skodol, A. E. (2012). Initial construction of a maladaptive personality trait model and inventory for DSM-5. *Psychological Medicine*, 42, 1879–1890.
- Lahey, B. B., Loeber, R., Burke, J. D., & Applegate, B. (2005). Predicting future antisocial personality disorder in males from a clinical assessment in childhood. *Journal of Consulting and Clinical Psychology*, 73, 389–399.
- Lenzenweger, M. F., Lane, M. C., Loranger, A. W., & Kessler, R. C. (2007). DSM-IV personality disorders in the National Comorbidity Survey Replication. *Biological Psychiatry*, 62, 553–564.
- Levenston, G. K., Patrick, C. J., Bradley, M. M., & Lang, P. J. (2000). The psychopath as observer: Emotion and attention in picture processing. *Journal of Abnormal Psychology*, 109, 373–385.
- Linehan, M. M., & Heard, H. L. (1999). Borderline personality disorder: Costs, course, and treatment outcomes. In N. E. Miller & K. M. Magruder (Eds.), *Cost-effectiveness of psychotherapy: A guide for practitioners, researchers, and policymakers* (pp. 291–305). London: Oxford University Press.
- Linehan, M. M. (1987). Dialectical behavior therapy for borderline personality disorder. *Bulletin of the Menninger Clinic*, 51, 261–276.
- Linehan, M. M., Heard, H. L., & Armstrong, H. E. (1993). Naturalistic follow-up of a behavioral treatment for chronically parasuicidal borderline patients. *Archives of General Psychiatry*, 50, 971–974.
- Loeber, R., & Hay, D. (1997). Key issues in the development of aggression and violence from childhood to early adulthood. *Annual Review of Psychology*, 48, 371–410.
- Lorber, M. F. (2004). Psychophysiology of aggression, psychopathy, and conduct problems: A meta-analysis. *Psychological Bulletin*, 130, 531–552.
- Lynch, T. R., Rosenthal, M. Z., Kosson, D. S., Cheavens, J. S., Lejuez, C. W., & Blair, R. J. R. (2006). Heightened sensitivity to facial expressions of emotion in borderline personality disorder. *Emotion*, 6, 647–655.
- Marshall, L. A., & Cooke, D. J. (1999). The childhood experiences of psychopaths: A retrospective study of familial and societal factors. *Journal of Personality Disorders*, 13, 211–225.
- McClure, M. M., Barch, D. M., Flory, J. D., Harvey, P. D., & Siever, L. J. (2008). Context processing in schizotypal personality disorder: Evidence of specificity of impairment to the schizophrenia spectrum. *Journal of Abnormal Psychology*, 117, 342–354.
- McCrae, R. R., & Costa, P. T. (1984). *Emerging lives, enduring dispositions: Personality in adulthood*. Boston: Little Brown.
- McCrae, R. R., & Costa, P. T., Jr. (1990). *Personality in adulthood*. New York: Guilford Press.
- McGlashan, T. H., Grilo, C. M., Sanislow, C. A., Ralevski, E., Morey, L. C., Gunderson, J. G., et al. (2005). Two-year prevalence and stability of individual criteria for schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *American Journal of Psychiatry*, 162, 883–889.
- McGlashan, T. H., Grilo, C. M., Skodol, A. E., Gunderson, J. G., Shea, M. T., Morey, L. C., et al. (2000). The collaborative longitudinal personality disorders study: Baseline axis I/II and II/II diagnostic co-occurrence. *Acta Psychiatrica Scandinavica*, 102, 256–264.
- Miller, J. D., Hoffman, B. J., Campbell, W. K., & Piskonis, P. A. (2008). An examination of the factor structure of Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, narcissistic personality disorder criteria: One or two factors? *Comprehensive Psychiatry*, 49, 141–145.
- Millon, T. (1996). *Disorders of personality: DSM-IV and beyond* (2nd ed.). New York: John Wiley & Sons.
- Minzenberg, M. J., Fan, J., New, A. S., Tang, C. Y., & Siever, L. J. (2008). Frontolimbic structural changes in borderline personality disorder. *Journal of Psychiatric Research*, 42, 727–733.
- Monet, G. (Producer). (1990). *Child of rage* [Documentary]. United States: Home Box Office.

- Morey, L. C., Hopwood, C. J., Markowitz, J. C., Gunderson, J. G., Grilo, C. M., McGlashan, T. H., et al. (2012). Comparison of alternative models for personality disorders, II: 6-, 8- and 10-year follow-up. *Psychological Medicine*, 42, 1705–1713.
- Morf, C. C., & Rhodewalt, F. (2001). Unraveling the paradoxes of narcissism: A dynamic self-regulatory processing model. *Psychological Inquiry*, 12, 177–196.
- Nelson, E. C., Heath, A. C., Madden, P. A. F., Cooper, M. L., Dinwiddie, S. H., Bucholz, K. K., et al. (2002). Association between self-reported childhood sexual abuse and adverse psychosocial outcomes: Results from a twin study. *Archives of General Psychiatry*, 59, 139–145.
- New, A. S., Hazlett, E. A., Buchsbaum, M. S., Goodman, M., Mitelman, S. A., Newmark, R., et al. (2007). Amygdala-prefrontal disconnection in borderline personality disorder. *Neuropsychopharmacology* 32, 1629–1640.
- Newman, J. P., Patterson, C. M., & Kosson, D. S. (1987). Response perseveration in psychopaths. *Journal of Abnormal Psychology*, 96, 145–149.
- Newton-Howes, G., Tyrer, P., & Johnson, T. (2006). Personality disorder and the outcome of depression: Meta-analysis of published studies. *British Journal of Psychiatry*, 188, 13–20.
- Newton-Howes, G., Tyrer, P., Anagnostakis, K., Cooper, S., Bowden-Jones, O., & Weaver, T. (2010). The prevalence of personality disorder, its comorbidity with mental state disorders, and its clinical significance in community mental health teams. *Social Psychiatry and Psychiatric Epidemiology*, 45, 453–460.
- Nigg, J. T., & Goldsmith, H. H. (1994). Genetics of personality disorders: Perspectives from personality and psychopathology research. *Psychological Bulletin*, 115, 346–380.
- Ogloff, J. R. P., Talevski, D., Lemphers, A., Wood, M., & Simmons, M. (2015). Co-occurring mental illness, substance use disorders and antisocial personality disorder among clients of forensic mental health services. *Psychiatric Rehabilitation Journal*, 38(1), 16–23.
- Oltmanns, T. F., & Powers, A. D. (2012). Gender and personality disorder. In T. A. Widiger (Ed.), *The Oxford handbook of personality disorders* (1st ed.). New York: Oxford University Press.
- Ozer, D. J., & Benet-Martinez, V. (2006). Personality and the prediction of consequential outcomes. *Annual Review of Psychology*, 57, 401–421.
- Patrick, C. J., Fowles, D. C., & Krueger, R. F. (2009). Triarchic conceptualization of psychopathy: Developmental origins of disinhibition, boldness, and meanness. *Development and Psychopathology*, 21, 913–938.
- Paulhus, D. L. (1998). Interpersonal and intrapsychic adaptiveness of trait self-enhancement: A mixed blessing? *Journal of Personality and Social Psychology*, 74, 1197–1208.
- Quirk, S. E., Berk, M., Chanan, A. M., Koivumaa-Honkanen, H., Brennan-Olsen, S. L., Pasco, J. A., & Williams, L. J. (2016). Population prevalence of personality disorder and associations with physical health comorbidities and health care service utilization: A review. *Personality Disorders: Theory, Research and Treatment*, 7(2), 136–146.
- Ragatz, L. L., Fremouw, W., & Baker, E. (2012). The psychological profile of white-collar offenders: Demographics, criminal thinking, psychopathic traits, and psychopathology. *Criminal Justice and Behavior*, 39, 978–997.
- Raine, A. (2006). Schizotypal personality: Neurodevelopmental and psychosocial trajectories. *Annual Review of Clinical Psychology*, 2, 291–326.
- Regier, D. A., Kuhl, E. A., & Kupfer, D. J. (2013). The DSM-5: Classification and criteria changes. *World Psychiatry*, 12, 92–98.
- Reich, J. (2000). The relationship of social phobia to avoidant personality disorder: A proposal to reclassify avoidant personality disorder based on clinical empirical findings. *European Psychiatry*, 15, 151–159.
- Reichborn-Kjennerud, T., Czajkowski, N., Torgersen, S., Neale, M. C., Orstavik, R. E., Tambs, K., et al. (2007). The relationship between avoidant personality disorder and social phobia: A population-base twin study. *American Journal of Psychiatry*, 164, 1722–1728.
- Reidy, D. E., Shelley-Tremblay, J. F., & Lilienfeld, S. O. (2011). Psychopathy, reactive aggression, and precarious proclamations: A review of behavioral, cognitive, and biological research. *Aggression and Violent Behavior*, 16, 512–524.
- Reiss, D., Heatherington, E. M., Plomin, R., Howe, G. W., Simmens, S. J., et al. (1995). Genetic questions for environmental studies: Differential parenting and psychopathology in adolescence. *Archives of General Psychiatry*, 52, 925–936.
- Ritter, K., Vater, A., Rusch, N., Schroder-Abe, M., Schutz, A., Fydrich, T., et al. (2014). Shame in patients with narcissistic personality disorder. *Psychiatry Research*, 215, 429–437.
- Roberts, B. W., Kuncel, N. R., Shiner, R., Caspi, A., & Goldberg, L. R. (2007). The power of personality: The comparative validity of personality traits, socioeconomic status, and cognitive ability for predicting important life outcomes. *Perspectives on Psychological Science*, 2, 313–345.
- Rutherford, M. J., Cacciola, J. S., & Alterman, A. I. (1999). Antisocial personality disorder and psychopathy in cocaine-dependent women. *American Journal of Psychiatry*, 156, 849–856.
- Samuel, D. B., & Widiger, T. A. (2008). A meta-analytic review of the relationships between the five-factor model and DSM-IV-TR personality disorders: A facet level analysis. *Clinical Psychology Review*, 28, 1326–1342.
- Samuel, D. B., Sanislow, C. A., Hopwood, C. J., Shea, M. T., Skodol, A. E., Morey, L. C., et al. (2013). Convergent and incremental predictive validity of clinician, self-report, and structured interview diagnoses for personality disorders over 5 years. *Journal of Consulting and Clinical Psychology*, 81, 650–659.
- Samuels, J., Eaton, W. W., Bienvenu, O. J., 3rd, Brown, C. H., Costa, P. T., Jr., & Nestadt, G. (2002). Prevalence and correlates of personality disorders in a community sample. *The British Journal of Psychiatry*, 180, 536–542.

- Sansone, R. A., & Sansone, L. A. (2011). Personality disorders: A nation-based perspective on prevalence. *Innovations in Clinical Neuroscience*, 8, 13–18.
- Shea, M. T., Stout, R., Gunderson, J., Morey, L. C., Grilo, C. M., McGlashan, T., et al. (2002). Short-term diagnostic stability of schizotypal, borderline, avoidant, and obsessive-compulsive personality disorders. *American Journal of Psychiatry*, 159, 2036–2041.
- Siever, L. J. (2000). S6. Genetics and neurobiology of personality disorders. *European Psychiatry*, 15, 54–57.
- Siever, L. J., & Davis, K. L. (2004). The pathophysiology of schizophrenia disorders: Perspectives from the spectrum. *American Journal of Psychiatry*, 161, 398–413.
- Silbersweig, D., Clarkin, J. F., Goldstein, M., Kernberg, O. F., Tiescher, O., Levy, K. N., et al. (2007). Failure of frontolimbic inhibitory function in the context of negative emotion in borderline personality disorder. *American Journal of Psychiatry*, 164, 1832–1841.
- Skodol, A. E., Oldham, J. M., Hyler, S. E., Stein, D. J., Hollander, E., Gallaher, P. E., et al. (1995). Patterns of anxiety and personality disorder comorbidity. *Journal of Psychiatric Research*, 29, 361–374.
- Smith, S. F., & Lilienfeld, S. O. (2013). Psychopathy in the workplace: The knowns and unknowns. *Aggression and Violent Behavior*, 18, 204–218.
- Soloff, P. H., Meltzer, C. C., Greer, P. J., Constantine, D., & Kelly, T. M. (2000). A fenfluramine-activated FDG-PET study of borderline personality disorder. *Biological Psychiatry*, 47, 540–547.
- Spitzer, R. L. (2002). *DSM-IV-TR casebook: A learning companion to the Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition, Text Revision*. Washington, D.C.: American Psychiatric Association.
- Spitzer, R. L., Gibbon, M., Skodol, A. E., Williams, J. B. W., & First, M. B. (Eds.). (1994). *DSM-IV casebook: A learning companion to the Diagnostic and Statistical Manual of Mental Disorders* (4th ed.). Washington, DC: American Psychiatric Press.
- Stone, M. H. (1993). *Abnormalities of personality. Within and beyond the realm of treatment*. New York: W. W. Norton.
- Sutker, P. B., & Adams, H. E. (2001). *Comprehensive handbook of psychopathology* (3rd ed.). New York: Kluwer Academic/Plenum.
- Taylor, S., Asmundson, G. J., & Jang, K. L. (2011). Etiology of obsessive-compulsive symptoms and obsessive-compulsive personality traits: Common genes, mostly different environments. *Depression and Anxiety*, 28, 863–869.
- Thomas, C. A., Turkheimer, E., & Oltmanns, T. F. (2003). Factorial structure of pathological personality as evaluated by peers. *Journal of Abnormal Psychology*, 112, 81–91.
- Tienari, P., Wynne, L. C., Laksy, K., Moring, J., Nieminen, P., Sorri, A., et al. (2003). Genetic boundaries of the schizophrenia spectrum: Evidence from the Finnish adoptive family study of schizophrenia. *American Journal of Psychiatry*, 160, 1587–1594.
- Torgersen, S., Lygren, S., Øien, P. A., Skre, I., Onstad, S., Edvardsen, J., et al. (2000). A twin study of personality disorders. *Comprehensive Psychiatry*, 41, 416–425.
- Torgersen, S., Myers, J., Reichborn-Kjennerud, T., Roysamb, E., Kubarych, T. S., & Kendler, K. S. (2012). The heritability of Cluster B personality disorders assessed both by personal interview and questionnaire. *Journal of Personality Disorders*, 26, 848–866.
- Trull, T. J., Jahng, S., Tomko, R. L., Wood, P. K., & Sher, K. J. (2010). Revised NESARC personality disorder diagnoses: gender, prevalence, and comorbidity with substance dependence disorders. *Journal of Personality Disorders*, 24, 412–426.
- Trull, T. J., Solhan, M. B., Tragesser, S. L., Jahng, S., Wood, P. K., Piasecki, T. M., et al. (2008). Affective instability: Measuring a core feature of borderline personality disorder with ecological momentary assessment. *Journal of Abnormal Psychology*, 117, 647–661.
- Tyrer, P. (2013). The classification of personality disorders in ICD-11: Implications for forensic psychiatry. *Criminal Behaviour and Mental Health*, 23, 1–5.
- Tyrer, P., & Bateman, A. W. (2004). Drug treatments for personality disorder. *Advances in Psychiatric Treatment*, 10.
- van Elst, L. T. (2003). Frontolimbic brain abnormalities in patients with borderline personality disorder: A volumetric magnetic resonance imaging study. *Biological Psychiatry*, 54, 163–171.
- Vazire, S., Naumann, L. P., Rentfrow, P. J., & Gosling, S. D. (2008). Portrait of a narcissist: Manifestations of narcissism in physical appearance. *Journal of Research in Personality*, 42, 1439–1447.
- Watts, A. L., Lilienfeld, S. O., Smith, S. F., Miller, J. D., Campbell, W. K., Waldman, I. D., et al. (2013). The double-edged sword of grandiose narcissism: Implications for successful and unsuccessful leadership among U.S. presidents. *Psychological Science*, 24, 2379–2389.
- Widiger, T. A., Frances, A., & Trull, T. J. (1987). A psychometric analysis of the social-interpersonal and cognitive-perceptual items for schizotypal personality disorder. *Archives of General Psychiatry*, 44, 741–745.
- Yang, Y., & Raine, A. (2009). Prefrontal structural and functional brain imaging findings in antisocial, violent, and psychopathic individuals: A meta-analysis. *Psychiatry Research: Neuroimaging*, 174, 81–88.
- Zanarini, M. C., Frankenburg, F. R., Reich, D. B., & Fitzmaurice, G. M. (2011). Attainment and stability of sustained symptomatic remission and recovery among patients with borderline personality disorder and axis II comparison subjects: A 16-year prospective follow-up study. *American Journal of Psychiatry*, 168, 1–8.
- Zanarini, M. C., Frankenburg, F. R., Hennen, J., Reich, D. B., & Silk, K. R. (2004). Axis I comorbidity in patients with borderline personality disorder: 6-year follow-up and prediction of time to remission. *American Journal of Psychiatry*, 161, 2108–2114.
- Zanarini, M. C., Skodol, A. E., Bender, D., Dolan, R., Sanislow, C., Schaefer, E., et al. (2000). The Collaborative Longitudinal Personality Disorders Study: Reliability of axis I and II diagnoses. *Journal of Personality Disorders*, 14, 291–299.

- Zeier, J. D., & Newman, J. P. (2013). Feature-based attention and conflict monitoring in criminal offenders: Interactive relations of psychopathy with anxiety and externalizing. *Journal of Abnormal Psychology, 122*, 797–806.
- Zimmerman, M., & Mattia, J. I. (1999). Differences between clinical and research practices in diagnosing borderline personality disorder. *American Journal of Psychiatry, 156*, 1570–1574.
- Zimmerman, M., Rothschild, L., & Chelminski, I. (2005). The prevalence of DSM-IV personality disorders in psychiatric outpatients. *American Journal of Psychiatry, 162*, 1911–1918.
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## CHAPTER 13

# Legal and ethical issues

### LEARNING OUTCOMES

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After studying this chapter, you should be able to:

- 13.1** differentiate the legal concepts of insanity and the various defences
  - 13.2** describe the issues surrounding fitness to stand trial
  - 13.3** delineate the conditions under which a person can be committed to a hospital under 'civil commitment', and the issues surrounding the rights to receive and refuse treatment
  - 13.4** describe the ethics surrounding psychological research and therapy.
-

## OPENING SCENARIO

David had been hearing voices for several days. Unable to drown them out with music or talking, he became more and more troubled. The voices were telling him that he was the one chosen by God to rid the world of evil. David went to the emergency room of the local hospital seeking relief. Instead of being admitted, David was given a prescription for the antipsychotic medication Haldol and sent on his way. Two days later, David took a loaded gun into a busy train station and began shooting. He killed two people and injured four others. When he was arrested, David told the police he was answering to God. His speaking was disorganised and hard to follow, and he expressed a number of paranoid beliefs.

David was found fit to stand trial because he understood the nature of his murder charge and he was able to give instructions to his criminal defence lawyer. At trial, David entered a plea of mental impairment. His lawyer arranged for David to be evaluated by a psychologist. The psychologist concluded that David had schizophrenia and that at the time of the crime he was unable to discern right from wrong (he thought his behaviour was the right thing to do since God was directing him) and unable to conform his behaviour to the requirements of the law. The Crown prosecutor did not dispute these findings and the court found David not liable for the murders by reason of mental impairment and made a forensic order for involuntary detainment at the Forensic Hospital in New South Wales. David was committed for an indeterminate period of time. Periodic evaluations and reviews by the Mental Health Review Tribunal would be conducted to determine whether David could be transferred to a less secure hospital or be granted community leave.

After 12 years in the hospital, David had done very well. He took his prescribed medication (Zyprexa), was never in a physical altercation with other people, participated in therapy, and actively engaged in vocational and leisure programs. David felt horribly remorseful for the crimes he had committed. He recognised that he had schizophrenia and that his illness would require treatment for the rest of his life. The treatment team all agreed that David could safely be housed in a less restrictive environment and that his schizophrenia was under control with the medication. They recommended that he be transferred to a medium security mental health unit so that he could begin transition towards community leave. The Crown prosecutor objected to David's custodial variation, after receiving complaints from members of the public who read about the upcoming transfer in the local newspaper. It was argued that David could stop taking his medications, become violent again and reoffend when released on leave into the community. At the tribunal hearing, it was agreed that the variation was premature, and the application was refused. David was to remain in the secure unit of the Forensic Hospital where he would continue to receive treatment.

### QUESTIONS

1. In your professional opinion, where would you consider would be the best place for David to be detained in light of his crimes (e.g., prison or elsewhere)?
2. In light of the particular facts of David's case, when and under what circumstances should he be released into the community, if ever?
3. Which members of the public do you foresee might object to the release of David into the community, and what effect might this have on decision makers?

## Introduction

We open our final chapter with the scenario about David because of the historical collaboration between the criminal justice system and mental health system to deny, although often subtly and perhaps unintentionally, a substantial proportion of the population in Australia and New Zealand their basic rights and liberties. With the best of intentions, judges, governing boards of hospitals, legal associations and professional mental health groups have worked over the years to protect society at large from the actions of people regarded as mentally ill and considered dangerous to themselves or to others. However, in so doing, based on a likely combination of misunderstanding and prejudice, they have denied many people their basic personal liberties by forcing them into treatment. Early laws relating to mandatory psychiatric treatment were written in legislation that allowed the operation of asylums or institutions that, while providing a level of care and safety, deprived people with mental illness of many of their personal liberties.

Until very recently (2013), laws in Australia have allowed forced psychiatric treatment if it was thought necessary to protect a person from serious harm, even if that person could competently refuse it.

People with a psychological disorder or mental impairment who have committed serious criminal offences, or who are alleged to have done so, are subject to **criminal commitment**. Criminal commitment is a term used in this chapter to describe, in a general sense, the procedure by which a court detains a person involuntarily in a mental health facility or forensic hospital. The purpose of criminal commitment is either to determine the person's fitness to stand trial, or after acquittal by reason of insanity, sometimes referred to as **mental impairment**, to detain the person in a mental health service, as in the opening scenario of David. **Civil commitment**, again used in a general sense, refers to the involuntary detention, treatment and care of a person who has, or is reasonably suspected of having, a mental illness and who is considered to be a risk to themselves or others. **Involuntary detention** can involve deprivation of a person's liberty, their compulsory treatment (medication) and placement in a mental health service or other authorised place (public hospital). In this chapter we provide an overview of these legal procedures. Then we turn to an examination of some important ethical issues as they relate to treatment and research. It is important to note that the law and professional ethics are closely interconnected.

In Australia, each of the six states and two territories has its own mental health system. Commonwealth laws provide funding for the provision of such services, and state and territory laws regulate the infrastructure of mental health services. This allows for some diversity in the provision of the care, treatment and control of persons who are mentally ill through hospitals and community care facilities in the various jurisdictions. For instance, while in all jurisdictions admission to a hospital or care facility can be on a voluntary or involuntary basis, some state mental health systems incorporate a specialist body, such as a **Mental Health Review Tribunal**, which reviews the involuntary detention and treatment of patients.

Each jurisdiction also has its own criminal justice system and criminal laws that define mental impairment as 'mental illness', an 'intellectual disability' or a 'specific neurological condition'. The criminal law is generally administered by individual jurisdictions including the states, territories and Commonwealth Government. Criminal law is largely a matter for the states. It is useful to note that in all jurisdictions (except NSW, Vic. and SA) the criminal law has been wholly codified (e.g., Criminal Code Acts). In New South Wales, Victoria and South Australia, the bulk of the criminal law is based on common law, but is partially expressed in legislation such as Crimes Acts that list the most common offences and fix their penalties. The mental health system often intersects with the criminal justice system when a person with a mental illness is accused of committing a serious crime.

In 2016, Queensland enacted the most recent review of mental health legislation in Australia. The *Mental Health Act 2016* (Qld) commenced 5 March 2017. The new Act will be referred to as a working example in this chapter, with references to other jurisdictions where relevant. Students are encouraged to look up similar references in mental health laws of other jurisdictions relevant to their practice.

Mental health law in New Zealand dates back to its beginnings as a modern state in 1840 with the signing of the Treaty of Waitangi between Maori tribal leaders and the British crown. Today, the *Mental Health (Compulsory Assessment and Treatment) Act 1992* provides for the compulsory treatment of people with a mental illness in New Zealand. The Ministry of Health is responsible for implementation of government policy (see Ministry of Health, 2012) and administration of mental health legislation. The Ministry provides funding for the provision of mental health and addiction services both in the community and inpatient services. Like in Australia, mental health law allows for both voluntary and involuntary treatment in the community and in mental health facilities.

## 13.1 Mental health and the criminal justice system

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**LEARNING OUTCOME 13.1** Differentiate the legal concepts of insanity and the various defences.

We begin by considering the role of psychiatry and psychology in the criminal justice system. Almost as early as the concept of *mens rea*, or 'guilty mind', and the rule 'No crime without an evil intent' had begun to be accepted in English common law, the concept of *insanity* was taken into consideration.

Broadly speaking, insanity refers to a disordered mind and a disordered mind may be regarded as unable to formulate and carry out a criminal purpose (Morse, 1992). In other words, a disordered mind cannot be a guilty mind and only a guilty mind can engender culpable actions.

It is important to note that insanity is a legal concept, not a psychological one. As such, its definition comes from court proceedings. In today's courts, judges and lawyers call on psychiatrists and clinical psychologists for assistance in dealing with criminal acts thought to result from the accused person's disordered mental state. Although the insanity defence was developed to protect people's rights, in practice, it often resulted in a greater denial of liberties than they would otherwise experience (see the clinical case of Bobby Veen).

## The insanity defence

The **insanity defence** requires a court to find that at the time of committing the illegal act, the accused's state of mind was such that they did not know what they were doing, or if they did, they did not know that it was wrong. The legal argument that an accused person should not be held responsible for an illegal act rests on whether the act is attributable to a psychological disorder that interferes with rationality or that results from some other excusing circumstance, such as not knowing right from wrong.

In Australia, the law of 'mental impairment' is the successor, in some jurisdictions, of the insanity defence. A second defence of insanity is **diminished responsibility**. According to Hemming (2008), both mental impairment and diminished responsibility defences rely on establishing, on the balance of probabilities, a *total incapacity* against one of three capacities for mental impairment and a *substantial incapacity* against one of three for diminished responsibility. Put simply, the three capacities are that the person did not know the nature and quality of the conduct; or that the conduct was wrong; or the person was unable to control their actions (see Criminal Code 1983 (NT) s 43C(1) Defence of mental impairment).

A staggering amount of material has been written on the old insanity defence even though it is seldom pleaded and even when pleaded is rarely successful. The defendant who successfully pleaded insanity 'faced a lifetime at Her Majesty's pleasure in an asylum for the criminally insane' (Hemming, 2008, p. 2). Today, Hemming states, 'a successful defence of mental impairment will lead to an accused person being liable to supervision or being released unconditionally' (p. 2).

Because an insanity defence was based on the accused's mental condition at the time the crime was committed, retrospective, often speculative judgement on the part of legal practitioners, judges, jurors and mental health professionals was required. Further, disagreement between defence and prosecution psychiatrists and psychologists was and continues to be the rule.

It is critically important to understand that psychological disorders and crime do not go hand in hand. That is, a person can have a diagnosis of a psychological disorder and be held fully responsible for a crime. For example, Donna Fitchett, a 51-year-old nurse, wife and mother living in Victoria with a confirmed diagnosis of a depressive disorder, killed her two sons in 2005. As a nurse, Fitchett obtained benzodiazepines to drug both her sons but the drugs did not take full effect so Fitchett suffocated one child before strangling the other. A jury found her guilty in 2008 and the judge sentenced her to 24 years imprisonment with a non-parole period fixed at 18 years. Her mental impairment plea was unsuccessful (see focus on discovery 13.3).

However, someone who has no mental illness at all can commit the most heinous or bizarre crime, despite our tendency to think someone must have been 'crazy' to commit such an act. Indeed, decades of social psychological research tell us that otherwise healthy people can commit horrendous criminal acts in the right circumstances or contexts. For example, 28-year-old Martin Bryant, Australia's most infamous killer, shot and killed 35 and injured another 23 people during a 'shooting spree' at the historic Port Arthur gaol site in Tasmania in 1996. The killing came to be known as the 'Port Arthur massacre' and led to nationwide gun control reforms banning semi-automatic firearms. Bryant was a quiet, socially isolated individual. The public was bamboozled as to why a young man raised by a loving family would commit such a monstrous crime. Bryant provided conflicting and confused accounts of what led him to commit the killings, leaving many to speculate that it could have been a desire for attention after allegedly telling a neighbour 'I'll do something that will make everyone remember me' (Bilowol & Davis, 2007). Bryant was found to be fit to stand trial and was allegedly

persuaded by his court-appointed lawyer and the prosecution to plead guilty. Prior to the guilty plea, prosecution and defence psychiatric reports assessed Bryant as having Asperger's syndrome, conduct disorder, attention-deficit hyperactivity disorder, or social and intellectual impairment but not mental illness. Bryant is currently serving 35 life terms plus a 1035-year cumulative sentence at Risdon Prison in Hobart with no prospect of release or parole. Media reports about Bryant were and continue to be scathing (see, for example, Blake, 2015).

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Martin Bryant is serving 35 life terms plus a cumulative 1035-year sentence at Risdon Prison in Hobart, Tasmania.



Some types of insanity defence, loosely defined, have been around since the seventh century BCE (Robinson, 1996). However, we will consider more recent legal precedents that set the stage for our current definitions of insanity and have shaped the development of the two main insanity pleas used today.

## Landmark cases and laws

Several court rulings and established principles bear on the problems of legal responsibility and psychological disorders. The following provides an overview of the most relevant.

### The M’Naghten rule

The **M’Naghten rule** was formulated in the aftermath of a murder trial in England in 1843. The defendant, Daniel M’Naghten, had set out to kill British Prime Minister Sir Robert Peel, but had mistakenly shot Peel’s secretary, Edward Drummond. At the time of his arrest, M’Naghten told police that he came to London to murder the prime minister because he claimed ‘the tories in my city follow and persecute me wherever I go and have destroyed my peace of mind. They do everything in their power to harass and persecute me; in fact they wish to murder me’ (M’Naghten’s Case, n.d.). At his trial, defence counsel introduced expert and lay witnesses who testified to M’Naghten’s delusions and that he suffered from acute insanity. The judge gave the jury an instruction regarding the accused’s lack of understanding upon commission of the act in question. The jury found him not guilty by reason of insanity.

Following the trial, a meeting at the House of Lords attended by 15 judges was held to determine the standards for the insanity defence. The judges ruled that juries must first be instructed that every man is presumed

sane and to possess a sufficient degree of reason to be responsible for his crimes. Then, to establish the insanity defence:

it must be clearly proved that, at the time of the committing of the act, the party accused was labouring under such a defect of reason, from disease of the mind, as not to know the nature and quality of the act he was doing; or if he did know it, that he did not know he was doing what was wrong. (*R v M'Naghten*, 1843, p. 7)

The M'Naghten rule has since been typically presented to juries as the standard used to determine whether an accused person, who raises the insanity defence, knew the difference between right and wrong at the time of doing the unlawful act. This is a cognitive-based test of knowledge where the accused (a) did not know *what* he or she was doing or (b) did not know the *act* he or she was doing, taking into consideration the relevant circumstances of the case.

In 1933, we see the application of the M'Naghten rule in a High Court of Australia decision in *R v Porter* (1933). The Court had to decide whether a father, Bertram Porter, was of such a mental condition at the time of murdering his infant son, by administering to him strychnine, as not to be criminally responsible. Dixon J emphasised that his 'state of mind must have been one of disease, disorder or disturbance'. If that is established, his state of mind must then have been 'of such a character as to prevent him from knowing the physical nature of the act he was doing or of knowing that what he was doing was wrong'. The M'Naghten rule continues to be applied in case law and is reflected in statute law.

Today in Australia, the criminal law sets out when a person is not criminally responsible for their acts or omissions based on a defence of insanity or mental impairment. Some jurisdictions rely on the common law. Other jurisdictions, like Queensland, rely on statutory criminal law that incorporates the M'Naghten standard.

For example, in Queensland, the *Criminal Code Act 1899* (Qld) s 27 Insanity states that a person is not criminally responsible for an act or omission if, at the time of doing the act or making the omission, the person is in such a state of mental disease or natural mental infirmity as to deprive the person of the capacity:

- to understand what the person is doing,
- to control their actions, or
- to know that they ought not commit the act or make the omission.

However, a state of mind resulting to any extent from intentional intoxication with alcohol or drugs is excluded.

It is rare for a defence lawyer to successfully plead the insanity or mental impairment defence in Australia because it is difficult to prove. The defence is based on numerous often conflicting psychological and psychiatric reports that tend to overwhelm juries. The plea of diminished responsibility is far less onerous but is only available in some jurisdictions.

Daniel M'Naghten had a mental disorder when he tried to kill the prime minister. His case helped to establish a legal definition for the insanity defence.



### FOCUS ON DISCOVERY 13.1

#### Mental impairment versus mental illness

On 29 January 2009, Arthur Phillip Freeman pulled over at the highest point of the West Gate Bridge in Melbourne and threw his four-year-old daughter Darcey over the edge. She fell 58 metres into the water below and died as a result of horrific impact injuries. Her two brothers, six-year-old Benjamin and two-year-old Jack, were both in the car at the time. It was to be Darcey's first day of primary school. ►

The nation was horrified by Freeman's actions. In the lead-up to his trial, it became known that he had been given reduced custody of his children in Family Court proceedings. This caused him a great deal of distress, which Freeman later described as 'having lost your children' (*R v Freeman*, 2011, p. 2). On the drive to West Gate Bridge, Freeman received two phone calls from his estranged wife, Ms Peta Barnes. In the first call, Freeman told her to say goodbye to her children, and in the second, he said she would never see her children again. In her victim impact statement, Ms Barnes described the impact of that horrific day:

Not a day goes by where I don't flashback to the emotions I felt when I was told by my ex-husband that I would never see my children again. The panic and fear these words set off inside me resonates within me even today. I feel them now in incidents of my daily life that would not have impacted me prior to Darcey's passing. I notice that I have heightened anxiety in everyday situations and have to manage myself carefully to control this. (*R v Freeman*, 2011, p. 2)

Leading up to his trial, Freeman was unable to describe the events of Darcey's death in detail. His defence lawyer argued that his client was not guilty of murder because of mental impairment. The trial was conducted on the premise that although Freeman was unable to remember what he had done, he did accept he was indeed the person responsible for Darcey's death. In his defence, psychiatrist Professor Graham Burrows argued that Freeman was 'suffering from severe depression and, as a result of that, [he] fell in a state of dissociation so that [his] acts were not conscious, voluntary and deliberate or intentional' (*R v Freeman*, 2011, pp. 3–4). However, other psychiatrists argued that he was 'suffering from mild to moderate depression and if there was any dissociation, it was not such as to have removed [your] capacity to act consciously, voluntarily and deliberately or intentionally' (*R v Freeman*, 2011, p. 4). The jury rejected the defence on mental impairment and he was convicted of murder. He was sentenced to life in prison with a non-parole period of 32 years.

This trial shows the difficulty of making a successful mental impairment defence based on conflicting psychiatric reports and is a vivid example of the critical difference between a mental illness and a defence of mental impairment.

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Arthur Freeman was sentenced to life in prison with a non-parole period of 32 years.



### QUESTIONS

1. What evidence would the jury require in their determination of whether Freeman was in fact mentally impaired when he was throwing his daughter off the West Gate Bridge?
2. What are the possible career implications for psychologists who provide expert testimony or reports that are favourable for the accused, in a highly publicised and emotive case?

### Diminished responsibility

Diminished responsibility originated in Scotland as a plea in mitigation of the death penalty for murder. It responded to the 'purely cognitive elements' of the M'Naghten insanity defence. It was then introduced in statute in England (see the *Homicide Act 1957* s 2(1)).

Today, four jurisdictions in Australia (Qld, NSW, ACT and NT) provide the partial defence known as diminished responsibility. Again, diminished responsibility is far easier to prove than the insanity defence. Consider Queensland as an example. If a person is charged with murder, section 304A of the *Criminal Code Act 1899* (the diminished responsibility provision) refers to an abnormality of mind (i.e., either arising from a condition of arrested or retarded development of mind or inherent causes or induced by disease or injury) that is found to substantially impair one or more of the capacities mentioned earlier in section 27 (above). If diminished responsibility is established, the charge is one of manslaughter only, not murder (as discussed in the clinical case of Bobby Veen, below).

Traditionally, juries have been tasked with deciding questions of unsoundness of mind and diminished responsibility in criminal trials. They can also be required to consider the question of an accused person's soundness of mind at the time of the trial (see s 645 Criminal Code Act) or whether the person is incapable of understanding the proceedings at trial (see s 613 Criminal Code Act). Prior to 1985, determinations of a mental health defence for mentally ill offenders was often exercised by the Mental Health Tribunal empowered by the *Mental Health Act 1974*. Today, the Mental Health Court is now able to decide questions of unsoundness of mind and diminished responsibility.

### The Mental Health Court

The Mental Health Court was established in Queensland by the Mental Health Act. The following provisions other than the criminal code apply to the *Mental Health Act 2016* (Qld). For a reference to be made to the Mental Health Court, there must be reasonable cause to believe that a person who is alleged to have committed an indictable (serious) offence:

- was of **unsound mind** (a state of mental disease of natural mental infirmity as described in s 27 of the Criminal Code) when the offence was allegedly committed
- is unfit for trial
- for a charge of murder, was of diminished responsibility when the offence was allegedly committed (s 110).

The matter of the person's mental condition relating to the offence may be referred to the Mental Health Court by:

- the person, their lawyer or the director of public prosecutions
- the chief psychiatrist or the director of forensic disability
- a Magistrate, Supreme or District Court.

Once a reference is made, court proceedings for the offence and any associated offences are suspended until the Mental Health Court has conducted a hearing and made a decision on the reference.

### Mental Health Court decisions

On the hearing of the reference, the Mental Health Court must decide whether the person who is the subject of the reference was of unsound mind at the time the offence was allegedly committed. Where the

offence is murder and the Court finds the person was not of unsound mind, it must decide whether the person was of diminished responsibility at the relevant time (s 116). The Court must not make one of the following decisions if there is a substantial dispute about whether the person committed the offence, unless the dispute is due to the person's mental condition or the operation of the Criminal Code in relation to s 304 (killing on provocation), s 304A (diminished responsibility) or s 304B (killing for preservation in an abusive domestic relationship).

Unless the Court has made a decision that the person was of unsound mind at the time of the alleged offence or of an alternative offence, or that there is no relevant dispute, it must decide whether the person is fit to stand trial and whether the unfitness is permanent (s 118).

#### **Finding of unsoundness of mind and fitness for trial**

If the Court decides that the person was of unsound mind when the offence was allegedly committed, the Court proceedings for the offence discontinue unless the person exercises the right to go to trial (s 119). The Court can make a forensic order allowing the person to be detained for treatment in an authorised mental health service or a high security unit. The Court can also make a treatment support order to remain in force until a final decision is made in the proceedings against the person for the offence.

#### **Finding of person fit for trial**

If the Court decides the person is fit for trial, it must order that proceedings for the offence be continued according to law (s 123). The Court may order that either the person remains in custody with or without bail, or is detained in an authorised mental health service until granted bail or brought before a court.

#### **Finding of unfitness for trial**

The Court may decide a person is unfit for trial on either a temporary or permanent basis. If the unfitness is deemed temporary, the proceedings for the offence are stayed until a tribunal, in Queensland the Mental Health Review Tribunal, reviews the matter and decides the person is fit for trial. If the Court finds a person to be permanently unfit for trial, the proceedings for the offence end (ss 121–122).

**Forensic orders** (*mental health* for those with a mental illness or disorder, or *disability* for those with an intellectual disability) or a **treatment support order** can be made by the Court (s 130). In deciding to make an order, the court must consider whether the person has a mental disorder or intellectual disability and:

- the relevant circumstances of the person
- the nature of the offence
- any victim impact statement produced to the court and
- the Chief Psychiatrist Policies relating to the management of persons on such orders.

*Forensic orders* are more restrictive of a person's rights and liberties than a treatment support order. The Court must make a forensic order if, because of the person's mental condition, it considers it necessary to protect the safety of the community from the risk of serious harm to other persons or property (s 134). Such orders can include conditions such as prohibiting contact with a victim of the relevant offence or the need to wear a tracking device (s 134). The Court can also make recommendations about intervention programs, such as drug and alcohol, anger management, counselling and sexual offender programs (s 136). However, the Court cannot impose a condition concerning the taking or dosage of particular medication. Where the matter before the Court involves a serious violent offence like murder, manslaughter or rape, the Court can impose a non-revocation period of up to 10 years on a forensic order.

There are two categories of forensic and treatment support orders: inpatient or community. Community orders are made if the person does not pose an unacceptable risk to the safety of the community. Forensic orders will state whether an authorised doctor or senior practitioner under the Forensic Disability Act can make future changes to any conditions or the nature or extent of treatment in the community (s 140).

A *treatment support order* is made if the Court considers a person poses a serious risk of harm to others or their property and needs treatment and care for a mental illness (s 143). The category of the order enables the Court to decide a 'least restrictive' approach to treatment. An *inpatient* order will be made only if the person's treatment and care needs and the safety and welfare of the person and others

cannot be met in the community (s 145). Unlike forensic orders, the Court (and the Mental Health Review Tribunal) does not set limits on the extent of community treatment under treatment support orders. This will be the responsibility of authorised doctors in accordance with the criteria set out in the Act. The Tribunal must also periodically review forensic orders to decide whether the order should be 'stepped down' to a treatment support order.

The criteria under the Act for the Mental Health Court (and the Tribunal) to make orders and the extent of treatment in the community can be summarised as follows:

- an inpatient category with no limited community treatment
- an inpatient category with limited community treatment (up to seven days) or
- a community category.

## Magistrates Courts

Magistrates Courts have the power under the *Mental Health Act 2016* (Qld) to discharge a person charged with a simple offence if it is reasonably satisfied, on the balance of probabilities, that the person was probably of unsound mind at the time of allegedly committing the offence or is unfit for trial (s 172). Alternatively, the Court can stay (delay) the proceedings if the unfitness for trial is considered temporary (i.e., the person is likely to be fit within six months). Magistrates may also order that a person be examined by an authorised doctor to decide if a treatment authority should be made for appropriate treatment and care (s 174).

In exceptional circumstances where the person is charged with a serious offence that creates a need to protect the community and the person appears to be or was of unsound mind at the time when the offence was allegedly committed, the Magistrates Court can refer the matter to the Mental Health Court.

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The Magistrates Court in Brisbane, Australia



## Highly publicised trials and public perception

As raised in the opening scenario about David, there was strong public sentiment in response to the potential for David to be released into the community should he be shifted to a less secure mental health facility. This type of public reaction is common in highly emotive and publicised crimes. It reflects the public's outrage based on misperceptions about the insanity defence and civil commitment processes. The public often believes that a person is 'getting away' with a crime when found not guilty by reason of insanity and that he or she will be released from the hospital in short order. In reality, many people who are committed to a mental hospital stay there longer than they would have stayed in prison had they been given a sentence. This is vividly illustrated in the clinical case of Bobby Veen.

### CLINICAL CASE

#### Bobby Veen

*Veen v R (No 2)* [1988] HCA 14

Robert 'Bobby' Veen was an Aboriginal man, one of the Stolen Generation. As infants, Bobby and later his sister Heather were adopted by a loving family and lived in Albury, Western Australia. When Bobby was 11 he was the victim of a crime that would upend his life. He later described the event as follows:

I was bundled in the car by three blokes and I was molested, brutally molested, you know. I lost a lot of faith then, me faith in religion and everything. That was the turning point, yeah, yeah. (Stewart, 2016)

Following the attack, Bobby started missing school, prompting the Aboriginal Protection Board to remove him from the Veen family and place him in the care of the notorious Kinchella Boys' Home. It was here that Bobby was subjected to abuse by staff and older boys during his eight-month stay. Bobby described the beatings, the rapes and molestations as being committed on everyone, and by the time he returned to his family, he was a changed person. His adoptive sister, Bernice, noted that, 'when he came back I remember Mum crying and saying how could they do that to a child?' (Stewart, 2016).

By the age of 14 Bobby had left the Veens and relocated to Sydney where he began work as a male prostitute. He became malnourished and a heavy drinker. In 1971, when almost 16, Veen stabbed his landlady while intoxicated at the boarding house where he lived. He was convicted of malicious wounding and committed to an institution. In 1975, at the age of 20, he committed his first murder. Again while intoxicated, he stabbed a male customer over 50 times with a kitchen knife when he refused to pay for sexual services over a weekend. The stabbing was triggered by the man saying 'you black bastards are all the same, always wanting handouts' (Stewart, 2016). He was charged with murder but convicted of manslaughter by a jury based on diminished responsibility. The judge gave Veen a life sentence based on a need to protect the community from his uncontrollable urges but this was reduced to 12 years' imprisonment on appeal.

He was released in January 1983 and committed a second murder later that same year. On this occasion, Veen was at a party, intoxicated and noticed two young boys around 11 and 12 in their underwear, drinking. He suspected they were being abused. His victim walked into the room and he said 'that's when I stabbed him, yeah ... I snapped, I just snapped' (Stewart, 2016). Veen stabbed the man repeatedly with a bread knife. Once again, Veen pleaded diminished responsibility and was sentenced to life imprisonment.

Veen remained in custody until June 2015 and became known as one of Australia's longest serving prisoners following a successful plea of diminished responsibility. He spent 42 years in prison. Speaking on behalf of his family, his sister Bernice said, 'we have felt he was kept there because nobody wanted to be the one responsible for signing off on Bobby Veen, releasing him back into society and having it happen for a third time ... it seems that the only reason he was released was because he was terminally ill' (Stewart, 2016). Veen died in March 2017, aged 62, less than two years after his release.

## 13.2 Fitness to stand trial

**LEARNING OUTCOME 13.2** Describe the issues surrounding fitness to stand trial.

The insanity or mental impairment defence concerns the accused person's mental state at the time of allegedly committing the crime. An important consideration before deciding what kind of defence to adopt is whether the accused person is competent to stand trial at all. In Australia and New Zealand's

criminal justice systems, **fitness to stand trial** is decided before it can be determined whether a person is responsible for the crime of which he or she is accused. In the Australian legal system, a person is 'presumed' to be mentally fit in order to be tried in a court of law.

At common law, a person who is considered 'unfit' to stand trial because of mental impairment cannot be tried. Justification for this approach includes that it avoids inaccurate verdicts by forcing a person who is incapable of answering for his or her actions to stand trial; it maintains the 'moral dignity' of the trial process; and it avoids unfairness by subjecting an unfit person to the trial process.

In the criminal law system, the common law test of unfitness to stand trial dates back to the significant case of *R v Pritchard* (1836). Pritchard, a man who was deaf and mute, was charged with bestiality, which at the time was punishable by death. Baron Alderson in that case set out what is now regarded as the legal basis for determining unfitness to stand trial, or the 'Pritchard criteria'. The relevant point was whether the defendant is 'of sufficient intellect to comprehend the course of proceedings on the trials, so as to make a proper defence — to know that he might challenge any of [the jury] to whom he may object — and to comprehend the details of the evidence...' Pritchard was ultimately found insane and detained under the then *Criminal Lunatics Act 1800*.

In 1958, the 'unfitness' test was said to involve six factors by the Victorian Supreme Court in *R v Presser* (1958). The requirements are *an understanding* of the nature of the charges and the court proceedings and the *ability* to challenge jurors, understand the evidence, decide what defence to offer and explain his or her version of the facts to counsel and the court. The unfitness to stand trial criteria as set out in the decision of Presser were incorporated into the (*Crimes Mental Impairment and Unfitness to Be Tried*) Act 1997 (Vic).

Another way to look at competency is that the courts do not want a person to be brought to trial *in absentia* ('not present'), which is a centuries-old principle of English common law that refers here to the person's mental state, not his or her physical presence. If, after examination, the person is deemed too mentally ill to participate meaningfully in a trial, the trial is delayed and the accused person is placed in an authorised mental health facility with the hope that the means of restoring adequate mental functioning can be found.

If a court fails to order a hearing when there is evidence that raises a reasonable doubt about competency to stand trial, or if it convicts a legally incompetent defendant, there is a violation of due process. As earlier indicated, the test to be applied is whether the defendant is able to consult adequately with his or her lawyer and whether he or she can understand the proceedings. However, analogous to the insanity defence, being deemed mentally ill does not necessarily mean that the person is incompetent to stand trial; a person with schizophrenia, for example, may still understand legal proceedings and be able to assist in his or her defence. In focus on discovery 13.1, we considered the case of Arthur Freeman who was found competent to stand trial despite clear evidence of psychological distress as stated in the psychological reports presented to the court.

Being judged incompetent to stand trial can have severe consequences for the individual. Bail may be denied, even if it would be routinely granted had the question of incompetency not been raised. The person is usually sent to an authorised mental health facility for examination and treatment in accordance with mental health law and may be detained until deemed fit to stand trial. In the meantime, the person may lose employment and undergo the trauma of being separated from family and friends and from familiar surroundings for months or even years, perhaps making his or her psychological state even worse and thus making it all the more difficult to show fitness or competency to stand trial.

#### CLINICAL CASE

##### Yolanda

Yolanda, a 51-year-old Aboriginal woman, was arrested after taking a box of doughnuts from the local supermarket. At the time of her arrest, she claimed she needed the doughnuts to feed the seven babies growing inside her. She said that convicted murderer Martin Bryant was the father of her soon-to-be-born children and that she would soon assume the position of Queen of the New Cities. When asked what the

New Cities were, she responded this was a new world order that would be in place following the alignment of the clouds with the planets Jupiter, Saturn and Venus. Yolanda's court-appointed lawyer immediately realised that Yolanda was not ready for trial; she asked for a stay of proceedings so that a psychologist could conduct an evaluation. Yolanda was diagnosed with schizophrenia and her thought disturbance was found to be so profound that she was not able to understand that she had been charged with a crime. Furthermore, she was unable to help her lawyer to prepare a defence. Instead, Yolanda viewed her lawyer as a threat to her unborn babies (she was not pregnant) and feared the lawyer would keep her from assuming her rightful position as queen. The judge declared that Yolanda was not fit to stand trial and ordered that she be sent to the local forensic mental health facility for treatment and further assessment.

At the facility, Yolanda was prescribed Olanzapine and her thinking became more coherent and organised after two months. One of the facility's psychologists worked with Yolanda, teaching her about the criminal justice system. She worked to help Yolanda understand what the charge of theft meant and what a defence counsel, Crown prosecutor, judge and jury were. At the end of three months, a different psychologist evaluated Yolanda and recommended that she now be considered competent to stand trial. Yolanda's public court-appointed defence lawyer came to the hospital and met with her to discuss the case. Yolanda was able to help her lawyer by telling her about her past hospitalisations and treatment history for schizophrenia. Yolanda realised she was not pregnant but still held onto beliefs about the New Cities. Still, Yolanda understood that she had stolen the doughnuts and this was why she had to go to court. At her next competency hearing, Yolanda was deemed fit to stand trial. Two months later, she went to court again. This time, she entered a plea of mental impairment. After a short trial, the judge accepted her plea and she returned to the forensic hospital. The treatment goals were now focused on helping Yolanda recover from schizophrenia, not on restoring her fitness to stand trial.

#### QUESTIONS

1. What are the critical factors that the court must have considered when making a determination of Yolanda's unfitness to stand trial?
2. How does a finding of 'unfitness to stand trial' affect the treatment goals for Yolanda as an alleged perpetrator of this type of offence?

## Victims of unlawful acts

Today, victims and their close relatives or another person who has been harmed by an unlawful act committed by a person who has a mental condition are given greater attention than in the past. In Queensland, for instance, the new Mental Health Act allows victims and other relevant persons to present a **victim impact statement** to the Mental Health Court and the Mental Health Review Tribunal. The impact statement is an application for the right to receive particular information about the relevant person through an **information notice**.

### Victim impact statements

A victim impact statement may be prepared by a victim or a close relative of a victim (e.g., spouses, siblings, children, parents and extended family members) and given to the prosecuting authority to present to the Mental Health Court when it is determining a person's soundness of mind or fitness to stand trial (s 162). Alternatively, a victim impact statement can be presented to the Mental Health Tribunal on review of a forensic order or treatment support order (s 742).

The statement may include:

- views about the risk the person represents to the victim or close relative or another person
- a request that when the Court makes its order, or the Tribunal reviews the order, a no-contact condition with the victim or other person is imposed.

A victim impact statement must not be disclosed to the person who is the subject of a forensic order or treatment support order unless at the request of the victim or close relative. If a request to disclose is made, disclosure can still be prohibited if it is considered harmful to the health and wellbeing of the

person of unsound mind. Victim impact statements may be given the weight considered appropriate when the Court or Tribunal makes decisions about a particular case.

### Information notices

A victim of an unlawful act or their close relative, or another person who has suffered harm because of the unlawful act and who has sufficient personal interest, may apply to the chief psychiatrist for an information notice (s 318). A chief psychiatrist protects the rights of patients in authorised mental health services. An information notice relates to a patient of an authorised mental health service who is subject to a forensic order or treatment support order. It may contain information that is considered relevant to the safety of victims such as when a forensic patient is granted community leave. Like victim impact statements, information notices are also subject to confidentiality requirements.

## 13.3 Civil commitment

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**LEARNING OUTCOME 13.3** Delineate the conditions under which a person can be committed to a hospital under 'civil commitment', and the issues surrounding the rights to receive and refuse treatment.

Historically, governments have had a duty to protect their citizens from harm. We take for granted the right and duty of government to set limits on our freedom for the sake of protecting us. Few drivers, for example, question the legitimacy of the state imposing limits on them by requiring seatbelts or by providing traffic signals that often make them stop when they would rather go. Government has a long-established right as well as an obligation to protect us both from ourselves — the *parens patriae*, 'power of the state' — and from others — the police power of the state. Civil commitment is one further exercise of these powers.

The philosophy underlying modern mental health laws is that, 'where possible, the voluntary treatment of mental illness should be regarded like the treatment of any other illness and should be part of the mainstream health system' (Caxton Legal Centre, 2011, p. 380). With that in mind, the main purpose of mental health law in Australia and New Zealand is to 'provide for the unique features of mental illness that cannot be catered for in other mainstream legislation. In particular, it provides for the treatment of mental illness when the person is incapable of consenting to treatment or is unreasonably objecting to treatment' (Caxton Legal Centre, 2011, p. 380). Before involuntary treatment can result, there are specific procedures for the involuntary detention and assessment, if necessary, of people who are reasonably believed to be mentally ill and in need of assistance (Caxton Legal Centre, 2011).

In Australia and New Zealand, Mental Health Acts in the various jurisdictions all define mental illness. This may vary from state to state. In Queensland, it is defined as a condition characterised by a clinically significant disturbance of thought, mood, perception or memory (s 10). The Mental Health Act specifically excludes a finding of mental illness merely because of certain affiliations or behaviour. For instance, if a person holds or refuses to hold a particular religious, cultural, philosophical or political belief or opinion; is a member of a particular racial group; has a particular sexual preference or orientation; engages in immoral or indecent conduct; takes drugs or alcohol; has an intellectual disability; engages in antisocial or illegal behaviour; is involved in family conflict; or has previously been treated for a mental illness or been involuntarily assessed or treated.

In all states, a person can be detained in an authorised mental health service or public hospital against his or her will if a judgement is made that he or she meets specific treatment criteria. This may vary from state to state but all states require the person has a mental illness and presents a risk to their own safety or welfare (the danger element) — that is, the person is suicidal or unable to provide for the basic physical needs of food, clothing and shelter — or to the safety of others. In Queensland, for example, the person's capacity to consent to be treated for the illness is an additional criteria. Civil commitment is supposed to last only as long as the person remains a safety risk to self or others.

A police or ambulance officer may take any person appearing to have a major disturbance in their mental capacity (i.e., caused by illness, disability, injury, intoxication or another reason), or who is at immediate risk of serious harm and appears to require urgent examination or treatment and care for the

disturbance, to an authorised mental health service or public hospital for examination (s 157B). The person may be detained for 6 to 12 hours while the emergency examination authority is being made, to determine the treatment and care needed, or to make a recommendation for assessment (s 157E).

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A police or ambulance officer may take a person into custody under the Mental Health Act.



In most states, a physician, not necessarily a psychiatrist, can make a recommendation for assessment that allows a person to be sent to an authorised mental health service and detained for some period of time, ranging from 24 hours to 72 hours (s 45). Detainment beyond this period requires the authorised doctor to make a treatment authority. The treatment authority authorises the involuntary treatment and care of a person for mental illness. It must state the grounds on which the authorised doctor is satisfied the treatment criteria apply to the person, the service responsible for the person's treatment and care, the category of the authority (inpatient or community) and any conditions considered necessary.

In Australia, a recent case has also shown that where a person is receiving inpatient treatment and care at an authorised mental health facility, the service owes the person a civil law duty of care when planning community leave. This duty extends to the patient's carer or support persons. In *Smith v Pennington* (2015) a young man was successful in establishing that the Local Health District had breached its duty of care when it failed to provide him and his parents with clear and explicit instructions prior to releasing him from a mental health unit where he was detained as an involuntary patient. He was diagnosed as a mentally disordered person following his second suicide attempt. It was well known to the mental health service and to his parents that alcohol and contact with his girlfriend were stressors that precipitated previous suicide attempts. The court case focused on the decision to grant him four days of leave from the mental health unit. He was released into his parents' care at the family home, where he unsuccessfully attempted suicide a third time by hanging himself. This resulted in permanent and catastrophic injuries (a hypoxic brain injury and Lance-Adams syndrome). His injuries left him wheelchair-bound

and in need of intensive caring. The main question was whether the hospital and its staff failed to give the plaintiff and his parents clear and explicit warnings and instructions about the key stressors and the precautions to be taken while he was on leave, prior to his release into the community. The Court found that he was owed a duty of care and this duty was breached, but that he failed to establish that the breach of duty caused his injuries. There was insufficient evidence to enable a conclusion to be drawn that any warnings about alcohol consumption or communication with the ex-girlfriend would have prevented him from attempting to take his own life. Essentially, he did not prove, on the balance of probabilities, that but for the breach of duty, he would not have sustained his injuries. Even if his parents had been given different warnings or information, it was not shown that in the circumstances they could have done more to protect their son from self-harm.

This case demonstrates the ongoing tension between the need to protect the rights of people with a mental illness, including their liberty and right to refuse medication, and the right of vulnerable people to be kept safe and receive appropriate treatment and care.

Civil commitment affects far more people than criminal commitment. It is beyond the scope of this text to examine in detail the variety of state civil commitment laws and regulations; each state has its own and they vary. Our aim is to present an overview that will provide a basic understanding of the issues and of current directions of change.

## **Preventive detention and problems in the prediction of dangerousness**

Unfortunately, there is a widespread public perception that people with psychological disorders account for a significant proportion of the violence that besets contemporary society, but this is not the case (Marie & Miles, 2008; Nielssen et al., 2011). In New South Wales, for instance, only 18 cases of stranger homicide by patients with a psychotic illness were reported between 1993 and 2002 (Nielssen, Westmore, Large, & Hayes, 2002). Moreover, about 90 percent of people diagnosed with psychotic disorders (primarily schizophrenia) are not violent (Swanson, Holzer, Ganju, & Jono, 1990). People with disorders such as schizophrenia or bipolar disorder — even allowing for their relatively small numbers — do not account for a large proportion of violent offences, especially when compared with people who abuse drugs or alcohol and people who are in their teens and 20s, are male and are poor (Corrigan & Watson, 2005; Douglas, Guy, & Hart, 2009; Fazel, Gulati, Linsell, Geddes, & Grann, 2009). The MacArthur Violence Risk Assessment Study, a large prospective study of violent behaviour among persons recently discharged from psychiatric hospitals in America, found that people with psychological disorders who did not also abuse substances were no more likely to engage in violence than were people without psychological disorders and substance abuse (Steadman et al., 1998).

When people with psychological disorders do act aggressively, it is usually against family members or friends, and the incidents tend to occur at home (Steadman et al., 1998). Indeed, stranger homicide by people with a psychological disorder is extremely rare (Nielssen et al., 2011). Another analysis from the MacArthur study found that people with psychological disorders reported more violent thoughts while in the hospital compared to people not in the hospital. However, these people were not necessarily more likely to actually be violent once they left the hospital (Grisso, Davis, Vesselinov, Appelbaum, & Monahan, 2000). Actual violent behaviour was found only among a subsample of people (e.g., those with a diagnosis of substance use disorder or those who had severe symptoms and persistent violent thoughts). Two meta-analyses of violence and psychological disorders focusing mostly on disorders that involve psychosis (schizophrenia spectrum disorders, bipolar disorder, depression with psychotic features) found that people with some type of psychotic disorder were slightly more likely to be aggressive, but this was particularly true when a person had the positive or disorganisation symptoms of schizophrenia (see the chapter on schizophrenia) or was also abusing drugs (Douglas et al., 2009; Fazel et al., 2009). Importantly, the increased risk for violence in people who also had a comorbid substance use disorder was similar to that found in people with a substance use disorder and no psychotic

disorder. This suggests that issues of substance abuse rather than psychotic disorders are the main contributory factors to violence. By and large, then, the general public is seldom affected by violence from people with psychological disorders, even though certain people with certain disorders can and will be violent.

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People with a psychological disorder are not necessarily more likely to be violent than people without a psychological disorder, contrary to the way movies often portray these people.



### **The prediction of violence**

The likelihood of committing a violent act is central to civil commitment, but is violence easily predicted? Early studies examining the accuracy of predictions that a person would commit a violent act found that mental health professionals were poor at making this judgement (e.g., Kozol, Boucher, & Garofalo, 1972; Monahan, 1973, 1976).

However, newer, empirically supported methods for assessing and predicting violence have been developed that identify and measure violence risk factors based on clinical judgement (e.g., Historical-Clinical-Risk Management-20, or HCR-20; Webster, Douglas, Eaves, & Hart, 1997) or a combination of clinical judgement and statistical algorithms (e.g., Violence Risk Appraisal Guide [VRAG]; Quinsey, Harris, Rice, & Cormier, 2006). These measures seem to work equally well, but even with these improved measures it remains a challenge to accurately predict future dangerousness and violence (Skeem & Monahan, 2011).

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Professor James Ogloff is the Director of the Centre for Forensic Behavioural Science at Swinburne University of Technology in Melbourne and specialises in the assessment and research of violence risk assessment in Australia.



Research suggests that violence prediction is most accurate under the following conditions (Campbell, Stefan, & Loder, 1994; Monahan, 1984; Monahan & Steadman, 1994; Steadman et al., 1998).

- If a person has been repeatedly violent in the recent past, it is reasonable to predict that he or she will be violent in the near future unless there have been major changes in the person's attitudes or environment.
- If violence is in the person's distant past and if it was a single but very serious act and if that person has been incarcerated for a period of time, then violence can be expected on release if there is reason to believe that the person's predetention personality and physical abilities have not changed and if the person is going to return to the same environment in which he or she was previously violent.
- Even with no history of violence, violence can be predicted if the person is judged to be on the brink of a violent act, for example, if the person is pointing a loaded gun at an occupied building.

### **Protection of the rights of people with psychological disorders**

In Australia and New Zealand, codes of professional conduct and codes of ethics require psychologists to keep their patients' private information in confidence unless the release of information is authorised, or compelled by law, or if it is in the public interest to do so. A breach of confidence occurs when confidential information is disclosed without appropriate authorisation or justification. Perhaps the most problematic exception to the duty to keep confidence is the public interest exception. As focus on discovery 13.2 demonstrates, the public interest exception is mostly derived from the common law or case law, which makes it difficult to specify with any certainty.

### The *Tarasoff* case — the duty to warn and to protect

The client's right to privacy and confidential communication is an important protection, but it is not absolute. Society has long stipulated certain conditions in which confidentiality in a relationship should not be maintained because of the harm that can befall others. A famous California court ruling in 1974 (*Tarasoff v Regents of the University of California*, 1974) described circumstances in which a therapist not only may but also *must*, at least in the US, breach the sanctity of a client's communication. First, we describe the facts of the case.

In the autumn of 1968, Prosenjit Poddar, a graduate student from India studying at the University of California at Berkeley, met Tatiana (Tanya) Tarasoff at a folk dancing class. They saw each other weekly during the fall and on New Year's Eve she kissed him. Poddar interpreted this act as a sign of formal engagement (as it might have been in India, where he was a member of the Harijan, or 'untouchable', caste). But Tanya told him that she was involved with other men and indicated that she did not wish to have an intimate relationship with him.

Poddar was depressed as a result of the rebuff, but he saw Tanya a few times during the spring (occasionally tape-recording their conversations in an effort to understand why she did not love him). Tanya left for Brazil in the summer and Poddar, at the urging of a friend, went to the student health facility, where a psychiatrist referred him to a psychologist for psychotherapy. When Tanya returned in October 1969, Poddar discontinued therapy. Based in part on Poddar's stated intention to purchase a gun, the psychologist notified the campus police, both orally and in writing, that Poddar was dangerous and should be taken to a community mental health centre for psychiatric commitment.

The campus police interviewed Poddar, who seemed rational and promised to stay away from Tanya. They released him and notified the health service. No further efforts at commitment were made because the supervising psychiatrist apparently decided that there was no need and, as a matter of confidentiality, requested that the letter to the police as well as certain therapy records be destroyed.

On 27 October, Poddar went to Tanya's home armed with a pellet gun and a kitchen knife. She refused to speak to him. He shot her with the pellet gun. She ran from the house; he pursued, caught and repeatedly and fatally stabbed her. Poddar was found guilty of voluntary manslaughter rather than first- or second-degree murder. The defence established with the aid of the expert testimony of three psychiatrists that Poddar's diminished mental capacity — schizophrenia — precluded the malice necessary for first- or second-degree murder. After his prison term, he returned to India, where, according to his own report, he is happily married (Schwitzgebel & Schwitzgebel, 1980, p. 205).

Under the privileged communication statute of California, the counselling centre psychologist properly breached the confidentiality of the professional relationship and took steps to have Poddar civilly committed, for he judged Poddar to be an imminent danger. Poddar had stated that he intended to purchase a gun and by his other words and actions he had convinced the therapist that he was desperate enough to harm Tarasoff. What the psychologist did not do, and what the court decided he should have done, was to warn the likely victim, Tanya Tarasoff, that her former friend had bought a gun and might use it against her. As stated by the California Supreme Court in *Tarasoff*: 'Once a therapist does in fact determine, or under applicable professional standards reasonably should have determined, that a patient poses a serious danger of violence to others, he bears a duty to exercise reasonable care to protect the foreseeable victims of that danger.' The *Tarasoff* ruling requires clinicians, in deciding when to violate confidentiality, to use the very imperfect skill of predicting dangerousness. Since the original ruling, it has been extended in several ways.

Prosenjit Poddar was convicted of manslaughter in the death of Tatiana Tarasoff. The court ruled that his therapist, who had become convinced Poddar might harm Tarasoff, should have warned her of the impending danger.



### Extending protection to foreseeable victims

A subsequent California court ruling (*Hedlund v Superior Court*, 1983) held by a bare majority that foreseeable victims include those in a close relationship with the identifiable victim. In this instance, a mother was hurt by a shotgun fired by the dangerous person and her seven-year-old son was present when the shooting took place. The boy later sued the psychologists for damages brought on by emotional trauma. Since a young child is likely to be in the company of his or her mother, the court concluded in *Hedlund* that the *Tarasoff* ruling extended to the boy.

### Extending protection further to potential victims

A 1983 decision of a federal circuit court in California (*Jablonski by Pahls v United States*, 1983) ruled that Veterans Administration psychiatrists should earlier have warned the murdered lover of an out-patient, Phillip Jablonski, that she was a foreseeable victim, even though the person had never made an explicit threat against her to the therapists. The reasoning was that Jablonski, having previously raped and otherwise harmed his wife, would likely direct his continuing 'violence ... against women very close to him' (p. 392).

The court also found the hospital psychiatrists negligent in not obtaining Jablonski's earlier medical records. These records showed a history of harmful violent behaviour, which, together with the threats his lover was complaining about, should have moved the hospital to institute emergency civil commitment. The court ruled that the failure to warn was a proximate or immediate cause of the woman's murder. Proper consideration of the medical records, said the judge, would have convinced the psychiatrists that Jablonski was a real danger to others and should be committed.

This broadening of the duty to warn and protect in the US has placed mental health professionals in California in an even more difficult predicament, for the potentially violent person need not even mention the specific person he or she may harm. It is up to the therapist to deduce who are possible victims, based on what he or she can learn of the person's past and present circumstances.

### Extending protection based on families' reports

In 2004, a California appeals court ruled that therapists have a duty to warn a possible victim if the threat is reported by a member of the patient's family (*Ewing v Goldstein*, 2004). In this case, a therapist learned about a threat not from his patient but from a family member of his patient. His patient revealed to his parents that he had thoughts of killing his ex-girlfriend's new boyfriend. The parents contacted the therapist about this threat. The therapist did not contact the new boyfriend, who was later killed by his patient. The parents of the boyfriend sued for wrongful death, saying the therapist should have warned their son. The court agreed and ruled that a close family member is in essence a part of the patient and thus a therapist does have a duty to warn potential victims if notified by a close family member of a patient.

### Extending protection to property

*Tarasoff* was further extended by a Vermont State Supreme Court ruling, *Peck v Counseling Service of Addison County* (1985), which held that a mental health practitioner has a duty to warn a third party if there is a danger of damage to property. The case involved a 29-year-old male who, after a heated argument with his father, told his therapist that he wanted to get back at his father and indicated that he might do so by burning down his father's barn. He proceeded to do just that. No people or animals were harmed in the fire; the barn housed no animals and was located 130 feet away from the parents' home. The court's conclusion that the therapist had a duty to warn was based on reasoning that arson is a violent act and therefore a lethal threat to people who may be in the vicinity of the fire.

In the United States, we can see an expanding willingness by some courts to find a duty of care to unidentifiable victims, provided the physician had some means available to prevent a tragedy. In Australia, these cases have yet to be given judicial affirmation. *Tarasoff* and other US cases therefore remain persuasive precedents only (not legally binding). Although the scope of 'public interest' remains vague and uncertain in Australia, the public benefit of being informed, in some circumstances, arguably outweighs the public interest in maintaining confidentiality. As the following English case demonstrates, in certain circumstances, there may be occasions when a health care professional receives information involving a patient that another's life is in real, serious and imminent danger and disclosure

is required. Since Australia and New Zealand share a similar legal system as England, the cases are far more persuasive.

*W v Edgell [1990] 1 All ER 855 (CA England)*

The plaintiff, W, had been convicted of manslaughter after killing five people. He was diagnosed as suffering from paranoid schizophrenia, which involved delusions of persecution by his neighbours. W was detained under a mental health order in a secure psychiatric hospital, the order being for an indefinite period. Some years later a Mental Health Review Tribunal considered his case and recommended that W be released to a less secure facility. This recommendation was based on a report by a Dr G, W's treating psychiatrist, that his condition was being controlled by medication and that he was suitable for transfer. The Home Secretary refused consent to the transfer and W appealed to the Tribunal for a review of the Home Secretary's decision. W's solicitor sought an opinion from Dr Edgell, an independent consultant. Dr Edgell's report was in fact unfavourable to W and he sent it to the solicitors believing it would be presented to the Tribunal. He was concerned, because he believed W was still dangerous and his outstanding interest in explosives and guns had not been adequately explored in Dr G's report. W's solicitors not wanting Dr Edgell's report to be seen by the Tribunal, withdrew W's application. Dr Edgell then felt compelled to send a copy of his report to the hospital where W was detained, the Home Secretary and the Department of Health and Social Security.

W sought an injunction to refrain further communication of the report, return of copies and damages for breach of confidence.

The Court dismissed W's application holding that Dr Edgell's duty to W to maintain confidentiality was overridden by his duty of care to bring his concerns to the proper authorities ... the risk to others must be 'real, immediate and serious' and W would not be released before further examination, the Court considered the circumstances of the case and held that the number of killings by W were enough to give rise to 'the gravest concern for the safety of the public'. (McIlwraith & Madden, 2014, p. 324)

The New Zealand case of *Furniss v Fitchett* (1958) has been regarded by most commentators as the precedent for an action in negligence for nervous shock resulting from a doctor's breach of the patient's confidence.

#### QUESTION

Discuss how the psychologist's ethical professional responsibilities to keep patient information confidential align with the law of confidentiality.

Being hospitalised against one's wishes is less likely today, in large part due to changes in health care that emphasise community-based or outpatient over inpatient care. In fact, today it is increasingly difficult to hospitalise a person who is in real need of a hospital stay, and this is also problematic. People with a psychological disorder such as schizophrenia or bipolar disorder may need hospital stays when symptoms become severe. Yet people in need of hospitalisation are often sent to a nursing home or boarding and care facility, which may offer little or substandard care. Worse, some people with psychological disorders in need of a hospital stay are likely to be sent to prisons instead of to a hospital, a topic we return to later.

Before we turn to a discussion of several issues and trends that revolve around the protections provided to those with psychological disorders, it is useful to consider the latest changes in mental health law in Australia.

The Queensland *Mental Health Act 2016* (s 5) provides principles to specifically protect the rights and interests of persons with mental illness. The overarching principle refers to the recognition of the same basic human rights that every person has in people with a mental illness, including a right to respect for their human worth and dignity as individuals. Significantly, there is a presumption of capacity in a person with mental illness to make decisions about their treatment and care. So, to the 'greatest extent practicable', the person is to be encouraged to take part in decisions affecting their life. Indeed, the Act requires decision makers to take into consideration certain matters, like the person's views, wishes and preferences about their treatment and care and the views of their support persons (family, carers and other support persons), as well as their privacy rights whenever decisions are to be made about the person.

Support and information to enable a person to exercise their rights under the Act is to be provided, again to the greatest extent practicable. This includes, for example, providing access to others who can help them express their views, wishes and preferences, and assistance to achieve their maximum physical, social, psychological and emotional potential, quality of life and self-reliance.

Other matters that must be taken into account include:

- a person's age-related, gender-related, religious and other special needs, including needs relating to hearing, visual or speech impairment; the unique cultural, communication and other needs of Aboriginal and Torres Strait Islander peoples and persons from other culturally and linguistically diverse backgrounds. For example:
  - providing treatment, care and support in a way that recognises and is consistent with their custom, mental health and social and emotional wellbeing, and is culturally and spiritually appropriate and respectful
  - assisting communication by providing an interpreter where practicable and appropriate in the circumstances.
- to the greatest extent practicable, the best interest of minors receiving treatment and care, including their specific needs, wellbeing and safety
- recovery-oriented services and reduction of stigma associated with mental illness
- that treatment and care be provided only if it is appropriate for promoting and maintaining the person's health and wellbeing
- a person's right to privacy and confidentiality of information.

### FOCUS ON DISCOVERY 13.3

#### Donna Fitchett

Donna Fitchett was the focus of popular media in Australia in late 2005, but for the worst reasons. Fitchett, a qualified nurse and devoted mother, murdered her two sons, aged 9 and 11, in their beds at the family home in Melbourne on 6 September 2005. She had carefully planned the killings, which involved giving the children Rohypnol, Valium and Normison; when this failed and the children began to wake up, she suffocated one child with a pillow and strangled the other with a stocking. Fitchett then made superficial cuts to her arms, neck and groin with a knife and ingested Rohypnol tablets to kill herself, she said. She was taken to hospital, discharged and then seen by the forensic medical officer the next morning. She was assessed as unfit to stand trial and certified for involuntary treatment with a diagnosis of depressive disorder. At the time of the killings, her marriage of 15 years was failing.

Prior to the murders, she had received counselling from a psychologist, Patra Antonis, on 37 occasions between 2004 and 2005. The counselling was in the context of her marital dissatisfaction. Near the time of killing her sons, she wrote a letter — that was never delivered — to Ms Antonis that said:

I told David it was over on the weekend because I'd already decided on my plan. Sadly I am too broken to go on. Today the boys will be given an overdose as I cannot and wouldn't ever abandon them. If I had real support from somewhere from someone who really cared it may have been different. Thomas and Matthew have had a wonderful childhood to date and I won't let anyone hurt them ever. They think we're going on an exciting trip today, but I've told them that they need to take some medicine so they won't get air sick. I'm not a coward nor am I crazy. I see this as my greatest act of love ... I know I was beyond help. I'm so sorry for the pain I will cause you but you gave me enormous peace by mirroring me. Our [sic] now want peace forever. (*R v Fitchett*, 2010, p. 3)

The issue at the trial was whether Fitchett was mentally impaired at the time she killed each boy, in particular, whether by reason of her mental impairment, she did not know that her conduct was wrong. The jury were not satisfied that the defence had been made out on the balance of probabilities. Two psychiatrists, Professor Mullen and Dr Sullivan, gave evidence to the Court on Fitchett's behalf. Whereas there was some disagreement as to the level of depression at the time of the killings (severe or moderate), both were of the opinion that by reason of her depression she could not comprehend that her conduct was wrong. The Crown prosecution called a consultant psychiatrist, Dr Skinner, to give evidence based on the transcript of the trial (but without conducting her own psychiatric examination). In Dr Skinner's opinion, Fitchett was suffering depression in the context of marital problems, which was probably mild to moderate in severity.

The jury returned a verdict of guilty to murder.

The judge accepted that Fitchett was a loving mother and that she was depressed at the time of killing her sons. However, she went on to say:

Nonetheless, you knew what you were doing, you contemplated it, planned it and wrote about it before you did it and then, when the boys did not succumb to the cocktails of drugs that you had given them, in respect of Thomas you strangled him and Matthew, you suffocated him and, consistently with the jury's verdict, you knew that what you were doing was wrong. Indeed, in your letter to Mrs Antonis, you wrote that you were not a coward, nor were you crazy and that this was your greatest act of love. That comment suggests to me that you understood the enormity of your actions and sought to justify them ... but in the greatest act of betrayal and in a profound breach of the trust which reposes in a parent, you robbed each of them of their precious lives because, in an act of unfathomable selfishness, you came to the view that, in your words, 'you couldn't and wouldn't ever abandon them'. Your crimes are truly appalling and offensive to civilised society and although you cling to the belief that your conduct is the result of your depressive illness, that does not obviate the fact that you have committed two chilling, callous murders. It follows that your crimes are properly to be regarded as very serious examples of the most serious offences. (*R v Fitchett*, 2010, pp. 6–7)

David Fitchett and other family and friends made victim impact statements that the Court took into consideration and to which the Judge responded, 'Your actions have resulted in unbearable, unforgiving pain for David Fitchett and for his family, and no sentence this Court can impose can restore life to your sons or peace to their father' (*R v Fitchett*, 2010, p. 8). Fitchett was sentenced to 34 years that culminated in an effective sentence of 27 years' imprisonment with a fixed non-parole period of 18 years. She was sent to a psychiatric unit within a prison, classified as a protected prisoner.

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Donna Fitchett was found guilty of murdering her two sons, aged 9 and 11.



### QUESTIONS

1. In light of the Tarasoff decision, consider what difference it would have made if Ms Antonis (Donna Fitchett's psychologist) had received the letter sometime prior to Fitchett committing the murders and disclosed it to another person.
2. Are there any legal or ethical implications if Ms Antonis said nothing after receiving the letter?
3. Consider the legal or ethical implications if Ms Antonis had received the letter prior to the crime being committed?

### Least restrictive alternative

Civil commitment or inpatient mental health care rest on 'presumed dangerousness', a condition that may vary depending on the circumstances. A person may be deemed to be a serious risk or dangerous if living alone in an apartment, but not dangerous if living in a residential treatment home and taking prescribed medications every day under medical supervision. The **least restrictive alternative** to freedom is to be provided when treating people with psychological disorders and protecting them from harming themselves and others. In general terms, mental health professionals have to provide the treatment that restricts the person's liberty to the least possible degree while remaining workable. It is unlawful to detain a person with a mental illness who is not a serious risk to self or others and who is capable of living in the community either on their own or with the support of willing and responsible family or friends. Of course, this principle has meaning only if society provides suitable residences, treatments and support services, which unfortunately does not happen as much as it needs to.

### Right to treatment

Another aspect of civil commitment that has come to the attention of the courts is the so-called right to treatment. If a person is deprived of liberty because he or she is mentally ill and is a danger to self or others, is the state not required to provide treatment to alleviate these problems?

In Australia and New Zealand, people's right to treatment and care for psychological disorders has historically been embedded in statute law and provided to everyone through a public health and later mental health care system. This can differ in other jurisdictions. In the United States, for example, the right to treatment was only extended to all people committed under civil law in a landmark 1972 case, *Wyatt v Stickney*. In that case, an Alabama federal court ruled that treatment is the only justification for the civil commitment of people with a psychological disorder to a psychiatric hospital. This ruling, upheld on appeal, is frequently cited in the US as ensuring the protection of people confined by civil commitment, at least to the extent that the state cannot simply 'put them away' without meeting minimal standards of care. In fact, when people with intellectual disability (as opposed to those deemed to be mentally ill) are released from an institution, health officials are not relieved of their constitutional duty to provide reasonable care and safety as well as appropriate training (*Thomas S v Flaherty*, 1990).

The *Wyatt* ruling was significant because previously the courts had asserted that it was beyond their competence to pass judgement on the care given to people with psychological disorders and they had assumed that mental health professionals possessed special and exclusive knowledge about psychopathology and its treatment. Repeated reports of abuses, however, gradually prodded the judicial system to rule on what went on within the walls of psychiatric hospitals. The *Wyatt* decision set forth very specific requirements for psychiatric hospitals — for example, dayrooms of at least 40 square feet, curtains or screens for privacy in multiperson bedrooms and no physical restraints except in emergency situations. The ruling also specified how many mental health professionals ought to be working in the hospital. When the *Wyatt* action was taken, Alabama state psychiatric hospitals averaged one physician per 2000 patients, an extreme situation. After *Wyatt*, there were to be at least two psychiatrists for every 250 patients.

Australia also has had its share of reports of abuse in mental health hospitals over the course of time. These reports and the cost of hospital care were instrumental in the changes towards community-based

care in the 1980s. By the 1990s, a series of government reports identified serious concerns with community-based care and human rights violations experienced by people with mental illness living in the community (see Human Rights and Equal Opportunity Commission, 1993). These fundamental changes to the way society provides treatment and care to people with mental illness are considered further in the section ‘Deinstitutionalisation, civil liberties and mental health’ below.

### Right to refuse treatment

Does a person committed under civil law have the right to refuse treatment or a particular kind of treatment? The answer is yes, but with qualifications.

The right of people with a mental illness to refuse medication is hotly debated in Australia and elsewhere. Psychiatrist E. Fuller Torrey (2014) asserts that because many people with a psychological disorder have no insight into their condition, they believe they do not need any treatment, and thus subject themselves and their loved ones to sometimes desperate and frightening situations by refusing medication or other modes of therapy, most of which involve hospitalisation. Torrey’s arguments plead for consideration of the costs of untreated psychological disorders, which are substantial (Torrey, 2014).

On the other hand, there are many arguments against forcing a person to take medications. The side effects of most antipsychotic drugs are often aversive and are sometimes harmful and irreversible in the long run. Notably, as many as one-third of people who take medications do not benefit from them.

Although there is inconsistency across jurisdictions, there is a trend towards granting even involuntarily committed people certain rights to refuse medication. In 2008, the United Nations Convention on the Rights of Persons with Disabilities demanded changes to laws that discriminate against people with disabilities, including mental illness. As a signatory to the Convention, Australia began reforms (since 2013) to its mental health acts. In Tasmania, South Australia, Western Australia and Queensland mental health laws have been amended giving people with mental illness ‘the same right to competently refuse psychiatric treatment as they do other medical treatment’ (Ryan, 2016). As mentioned above, the Queensland Act specifically deals with this issue by stating in its principles that the provision of treatment must be provided ‘only if it is appropriate for promoting and maintaining the person’s health and wellbeing’ (see s 5(1)).

In other jurisdictions (ACT, NSW and Vic.) forced psychiatric treatment can be administered, even if competently refused, if it is thought necessary to protect a person from serious harm. However, clinical psychiatrist Christopher Ryan states that in these jurisdictions:

There are a series of legal hoops to jump through that should mean this will only happen rarely. In NSW, for example, doctors must now make ‘every effort reasonably practicable’ to obtain a mentally ill person’s consent to treatment. This means doctors have to try their utmost to negotiate a way forward without resorting to involuntary treatment. (Ryan, 2016)

Ryan says that Australia is leading the world in its efforts to give everyone, including people with mental illness, the same rights. He has remarked that the United Kingdom and New Zealand are yet to make similar changes and ‘the United States has not even ratified the convention’ (Ryan, 2016).

Whereas the US relies on court interpretations of constitutional protections instead of legislation, there are some shared underlying concepts. In an extension of the least-restrictive-treatment principle, for instance, a US court ruled that the government cannot force antipsychotic drugs on a person only on the supposition that at some future time he or she might become dangerous (*United States v Charters*). The threat to the public safety has to be clear and imminent to justify the risks and restrictions that such medications pose and it must be shown that less intrusive intervention will not likely reduce impending danger to others. In other words, forcible medication necessarily restricts liberty in addition to whatever physical risks it could bring; there has to be a very good reason to deprive a person of liberty and privacy via such intrusive measures. For example, a ruling in favour of forcible medication was made in a case of a person who had been threatening to assassinate the president of the United States. He posed a threat to his own safety and he could be shown by clear and convincing evidence to be seriously mentally impaired (*Dautremont v Broadlawns Hospital*, 1987).

## Deinstitutionalisation, civil liberties and mental health

Court rulings such as *Smith v Pennington* put mental health professionals on notice to be careful about keeping people in psychiatric wards against their will, and emphasised that they also owe their patients a duty of care that extends to preparations for the patient's release into the community. This includes careful attention to the specific treatment needs of people with psychological disorders and the provision of critical information to carers or support persons.

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Civil commitment supposedly requires that the person be dangerous. But in actual practice, the decision to commit can be based on a judgement of severe disability, as in the case of some people who are homeless.



Between 1955 and the mid-1960s, following shocking reports of utter neglect of many state institutions and increasing budget pressures, the policy referred to as *deinstitutionalisation* started taking shape, discharging as many people as possible from hospitals and discouraging admissions.

In 1983, the Richmond Inquiry published a report into Health Services for the Psychiatrically Ill and Developmentally Disabled. The major focus was into the services provided by the public sector, including state-run psychiatric hospitals, public hospitals and community health programs. It argued for a more centralised and integrated model of care and support, with less emphasis on the dominant hospital-based model of care. Hospitals were targeted for closure with funding and support transferring to community-based services (i.e., community support services, home services, rehabilitation programs, training programs and residential care facilities). By 2000, all states and territories in Australia had transferred the management of public health services to the mainstream health system. It has been estimated that between 1992 and 2002, the number of beds in standalone psychiatric institutions fell by 60 percent (Commonwealth Department of Health and Ageing, 2004). Subsequently, the demand for acute beds in Australia is found to have grossly outweighed availability (Shelter NSW, 2002). In real terms, the additional 558 beds in the community care units represented only about a quarter of the 2285 long-stay beds that were reduced in psychiatric hospitals in Australia. In New South Wales, it was estimated that the total number of psychiatric beds in the state 'have declined from 12 000 in 1970 to approximately 2100' by 2002 (Shelter NSW, 2002).

According to Crowley-Cyr (2010), despite the system overhaul, some 20 years later, reports prepared by the Mental Health Council of Australia (MHCA) documented experiences of fundamental service failures, including deficiencies in community-based mental care. Unfortunately, there are woefully inadequate resources for such treatment in the community. Some effective programs were described in the chapter on schizophrenia, but these are very much the exception, not the rule. The state of affairs in cities is an unrelenting crisis for the hundreds of thousands of people with psychological disorders who have been released from hospitals without adequate community services to help them. Many people discharged from hospitals are eligible for benefits from the government including disability pensions, but a large number are not receiving this assistance. Rates of homelessness have soared among those with psychological disorders and homeless persons do not have fixed addresses and need help in establishing eligibility and residency for the purpose of receiving benefits. The state of homelessness undoubtedly exacerbates the suffering of people with psychological disorders.

Deinstitutionalisation may be a misnomer. *Transinstitutionalisation* may be more apt, for declines in the number of psychiatric hospital beds have occasioned increases in the presence of people with psychological disorders in nursing homes, the mental health departments of non-psychiatric hospitals and, most sadly, prisons and immigration detention centres (Crowley-Cyr, 2005; Kiesler, 1991; Torrey, 2014). These settings are by and large not equipped to handle the particular needs of people with psychological disorders.

Indeed, prisons and immigration centres have become the major treatment sites in Australia for people with psychological disorders in the twenty-first century. As Crowley-Cyr (2010) states:

Australian studies of prisoner populations . . . reveal a history of high prevalence of mental disorders and a lack of mental health services within prisons. A largescale study in 2005 examined two prisoner populations in New South Wales: 1) new receptions to the correctional system and 2) sentenced prisoners. The study found 46% of the screened reception prisoners and 38% of sentenced prisoners suffered from a mental illness. Further, women were found to have higher levels of psychiatric morbidity than men. (p. 203)

Nowadays, decommissioned heritage-listed ‘asylums’ act as tourist attractions for people interested in ghost tours. Speaking of the US position, William Wayne J in *Ruiz v Johnson* (1999) commented:

It is deplorable and outrageous that this state’s prisons appear to have become a repository for a great number of its mentally ill citizens. Persons who, with psychiatric care, could fit well into society, are instead locked away, to become wards of the state’s penal system. Then, in a tragically ironic twist, they may be confined in conditions that nurture, rather than abate, their psychoses.

Clearly, we must do more.

## 13.4 Ethical dilemmas in therapy and research

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**LEARNING OUTCOME 13.4** Describe the ethics surrounding psychological research and therapy.

The issues reviewed thus far place legal limits on the activities of mental health professionals. These legal constraints are important, for laws are one of society’s strongest means of encouraging all of us to behave in certain ways. Mental health professionals also have ethical constraints. Codes of ethics are designed to provide an ideal to review moral issues of right and wrong that may or may not be reflected in the law. All professional groups promulgate ‘shoulds’. These ethics guidelines describe what therapists and researchers should do with their patients, clients and research participants. Courts have also ruled on some of these questions. Most of the time what we believe is unethical is also illegal, but sometimes existing laws are in conflict with our moral sense of right and wrong. The Australian Psychological Society (APS, 2007) and New Zealand Psychological Society (NZPS, 2002) both publish a code of ethics that includes the ethical standards that constrain research and practice in psychology. We now examine the ethics of making psychological inquiries into and interventions in the lives of other human beings. The information in the following sections has been obtained from the APS *Code of Ethics* (2007),

*Code of Ethics For Psychologists Working in Aotearoa/New Zealand* (2002), the *Australian National Statement on Ethical Conduct in Human Research* (2007) and others where stated.

## Ethical restraints on research

The training of scientists equips them to pose interesting questions and to design research that is as free of confounds as possible. Scientists have no special qualifications, however, for deciding whether a particular line of research with people should be followed. Society needs knowledge and a scientist has a right in a democracy to seek that knowledge. However, the ordinary citizens who participate in experiments must be protected from unnecessary harm, risk, humiliation and invasion of privacy.

Perhaps the most reprehensible ethical insensitivity was evidenced in the brutal experiments conducted by German physicians on concentration camp prisoners during World War II. One experiment, for example, investigated how long people lived when their heads were bashed repeatedly with a heavy stick. Clearly, such actions violate our sense of decency and morality. The Nuremberg Trials, conducted by the Allies following the war, brought these and other barbarisms to light and meted out severe punishment (including the death penalty) to some of the soldiers, physicians and Nazi officials who had engaged in or contributed to such actions, even when they claimed that they had merely been following orders.

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Defendants at the Nuremberg Trials



It would be reassuring to be able to say that such gross violations of human decency take place only during incredible and cruel epochs such as the Third Reich, but unfortunately this is not the case. Spurred on by a blind enthusiasm for their work, researchers in the United States and other countries have sometimes dealt with human participants in reproachable ways.

For example, one experiment conducted after World War II compared penicillin with a placebo as a treatment to prevent rheumatic fever. Even though penicillin had already been acknowledged as the drug of choice for people with a streptococcal respiratory infection in order to protect them from later contracting rheumatic fever, placebos were administered to 109 service personnel without their knowledge or permission. More participants received penicillin than received the placebo, but three members

of the control group contracted serious illnesses — two had rheumatic fever and one had acute nephritis, a kidney disease. None of those who had received penicillin contracted such illnesses (Beecher, 1966).

Half a century later, in January 1994, spurred on by Eileen Welsome, a journalist who won a Pulitzer Prize for her investigative reporting on the issue, the United States Energy Department began to publicise numerous experiments conducted in the 1950s through the 1970s that had exposed hundreds of people — usually without their informed consent or prior knowledge — to harmful doses of radiation. Particular concern was expressed because the overwhelming majority were people of low socioeconomic status, members of ethnic minorities, people with intellectual disabilities, nursing home patients or prisoners. The scientists, for the most part supported in their research with federal funds, understood that the risks were great, even though relatively little was known about the harmful effects of radiation at the time. Some of these experiments involved giving women in the third trimester of pregnancy a radioactive tonic to determine safe levels of exposure and irradiating the testicles of prisoners to find out the degree of radiation that service personnel could endure without negative effects on sperm production.

Responding to the many instances of harm inflicted on research participants, several international codes of ethics for the conduct of scientific research have been developed — the *Nuremberg Code* formulated in 1947 in the aftermath of the Nazi war-crime trials, the 1964 *Declaration of Helsinki* and statements from the British Medical Research Council.

In 1966, the National Health and Medical Research Council (NHMRC) in Australia issued the *Statement on Human Experimentation* that expressly drew on the Helsinki Declaration. In 1976, a Supplementary Note 1 was added to include peer group assessment of experiments, meaning that all human research was subject to review by a Human Research Ethics Committee. Currently, there are a number of codes, statements and guidelines that govern human and animal research in Australia. These include, but are not limited to:

- *The Australian Code for the Responsible Conduct of Research* (2007)
- *The National Statement of Ethical Conduct in Human Research* (2007)
- *Values and Ethics: Guidelines for Ethical Conduct in Aboriginal and Torres Strait Islander Health Research* (2003)
- *The Australian Code for the Care and Use of Animals for Scientific Purposes 8th edition* (2013).

These various codes and principles are continually being re-evaluated and revised as new challenges are posed to the research community.

In New Zealand, there are a number of committees and regulatory bodies that govern ethical and best practices of health and disability research. These include: the Health Research Council of New Zealand, the National Ethics Advisory Committee, the Ethics Committee on Assisted Reproductive Technology and the National Animal Ethics Advisory Committee. For psychologists working in New Zealand, all research conducted in Maori communities should be guided by the three principles of the Treaty of Waitangi (6 February 1840) — partnership, participation and protection.

For more than four decades, the proposals of behavioural researchers, many of whom conduct experiments related to psychopathology, have been reviewed for safety and general ethical propriety by institutional review boards in hospitals, universities and research institutes. Such committees — and this is significant — comprise not only behavioural scientists but also citizens from the community. They are able to block any research proposal and they can require questionable aspects to be modified if in their collective judgement the research would put participants at too great a risk. Such committees now also pass judgement on the scientific merits of proposals, the rationale being that it is not ethical to recruit participants for studies that will not yield valid data (Capron, 1999). Beginning in 2000, universities and other research institutions have been required to certify researchers on the basis of special coursework and examinations concerning research ethics. Researchers who receive funds from federal agencies, such as the National Institute of Mental Health, are also required to receive specialised training in research ethics.

## Informed consent

A core component of ethical research is **informed consent**. The investigator must provide enough information to enable people to decide whether they want to be in a study. Researchers must describe the study

clearly, including any risks involved. Researchers should disclose even minor risks that could occur from a study, including emotional distress from answering personal questions or side effects from drugs. There must be no coercion in obtaining informed consent. Participants must understand that they have every right not to take part in the study or to withdraw from the study at any point, without any fear of penalty. For example, a psychologist might want to determine whether imagery helps students to associate one word with another. One group of students might be asked to associate pairs of words in their minds by generating a wacky image connecting the two, such as a cat riding on a bicycle. Current procedure allows prospective participants to decide that the experiment is likely to be boring and to decline to participate.

A central issue is that potential participants must be able to understand the study and associated risks. What if the prospective participant is an adult with a profound intellectual disability, unable to understand fully what is being asked? In clinical and research settings, researchers must ascertain that people are not having trouble understanding the study. In the 2007 *Australian National Statement on Ethical Conduct in Human Research*, ethical considerations are given for specific participants. These include but are not limited to: children and young people; people highly dependent on medical care who may be unable to give consent; and people with a cognitive impairment, an intellectual disability or a mental illness. The statement describes how the distinct vulnerabilities of these specific populations should be taken into account when obtaining consent. For example, for those with a cognitive impairment, intellectual disability or mental illness, who do not have the capacity to consent, informed consent is sought from the person's guardian or any person or organisation authorised by law. Nevertheless, the researcher is required, *as far as practically possible*, to explain the purposes of the research to the participant and what participation involves.

Still, as with the right to refuse treatment, there is recognition that people with a psychological disorder are not necessarily incapable of giving informed consent (Appelbaum & Gutheil, 1991). For example, although people with schizophrenia may do more poorly than people without schizophrenia on tests designed to assess decision-making skills, people with schizophrenia can give informed consent if a more detailed procedure describing a study is included — for example, one that describes what they will be asked to do and what they will see, and explains that their participation is voluntary and that it in no way will impact their treatment (Carpenter et al., 2000; Wirshing, Wirshing, Marder, Liberman, & Mintz, 1998).

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Informed consent must be obtained for research.



The issue of informed consent is also of concern to researchers and clinicians who work with people with Alzheimer's disease. As with schizophrenia, having an Alzheimer's diagnosis does not necessarily mean a person cannot provide informed consent (Marson, Huthwaite, & Hebert, 2004). Measures have been developed to assess capacity for consent in this population; as the number of people over age 65 continues to increase, this will continue to be an area of active research (Marson, 2001).

These results point to the importance of examining each person individually for ability to give informed consent, rather than assuming that a person hospitalised for schizophrenia or Alzheimer's disease is unable to do so.

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What a person tells their therapist is confidential, although in certain situations confidentiality may be broken.



## Confidentiality and ethical limits

When people consult a psychologist, they are assured by professional ethics codes that what goes on in the session will remain confidential. **Confidentiality** is defined in the APS *Code of Ethics* as the protection of information obtained during their provision of psychological services. It should not be released or discussed with third parties without the consent of the client, or a person with legal authority to act on behalf of the client unless it falls under the following circumstances or *limits of confidentiality*:

- where there is a legal obligation to do so
- if there is an immediate and specified risk of harm to an identifiable person or persons that can be averted only by disclosing information (recall focus on discovery 13.2 on *Tarasoff* and other case laws)
- when consulting colleagues, or in the course of supervision or professional training, provided the psychologist conceals the identity of clients and associated parties involved; or obtains the client's consent and gives prior notice to the recipients of the information that they are required to preserve the client's privacy and obtains an undertaking from the recipients of the information that they will preserve the client's privacy.

In the New Zealand *Code of Ethics*, limits to confidentiality include:

- *diminished capacity* in which case consent is obtained by a legally authorised person
- *children/young persons* and that significant health, safety and/or relationship issues may override confidentiality
- *urgent need* in which an urgent situation prevents the ability to gain consent to disclose in time to prevent harm or injury to the person, persons, family, whanau or community group
- *legal requirements* where a psychologist is compelled by law to disclose information given by a client or research participant
- *client or public safety* where a psychologist believes that non-disclosure may endanger a client, research participant or another person but is denied permission to disclose, the psychologist exercises professional judgement in deciding whether to breach confidentiality or not.

#### RESEARCH EXAMPLE

##### The Centre for Forensic Behavioural Science

The Centre for Forensic Behavioural Science located in Victoria is Australasia's leading centre for research in forensic mental health and forensic behavioural science. Some of the research conducted at the centre has been on risk assessment, specifically on validating North American risk assessment instruments to assess the prediction of violence in Australia and the Australian prison system. The centre has also been instrumental in the development and validation of a number of novel risk assessment instruments used to predict and assess the risk of imminent violence. Examples include the Dynamic Appraisal of Situational Aggression (Ogloff & Daffern, 2006) designed to identify and respond effectively to risks of future aggression from a psychiatric inpatient cohort, and the Stalking Risk Profile (MacKenzie et al., 2009) designed to assess the risk of stalking behaviour.

An area of important research conducted by the Centre for Forensic Behavioural Science is in the prediction of violence in young offenders, particularly those of Aboriginal or Torres Strait Islander ethnicity. As Shepherd and Strand (2016) discuss, cross-cultural youth psychopathology is an area that is inadequately researched even though Indigenous Australians are heavily over-represented in Australia's juvenile and adult criminal justice systems.

##### QUESTION

Why is it important to validate international risk assessment instruments on the Australian population?

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## SUMMARY

### 13.1 Differentiate the legal concepts of insanity and the various defences.

*Insanity* is a legal term, not a mental health term. Meeting the legal definition is not necessarily the same thing as having a diagnosable psychological disorder. The insanity defence — or mental impairment, as referred to in some jurisdictions — is the legal argument that a person is not criminally responsible for an act or omission if, at the time of doing the act or making the omission, the person is in such a state of mental disease or natural mental infirmity as to deprive the person of the capacity to: (a) understand what the person is doing; b) control their actions; or (c) to know that they ought not commit the act or make the omission. The defence clearly states that a state of mind resulting to any extent from intentional intoxication with alcohol or drugs is excluded. The M’Naghten rule specified that a person could not distinguish right from wrong at the time of the crime because of the person’s psychological disorder. It continues to be applied in case law and is reflected in statute law in Australia. Diminished responsibility is a partial defence and responds to the ‘purely cognitive elements’ of the M’Naghten insanity defence. Diminished responsibility can be used in four jurisdictions of Australia (Qld, NSW, ACT and NT) when an individual is charged with murder and is proved to have an abnormality of mind that is found to substantially impair one or more of the capacities of the insanity defence. If established, the charge is one of manslaughter only, not murder. However, as discussed in the clinical case of Bobby Veen, such a defence may in fact result in a greater denial of liberties than would be otherwise experienced.

### 13.2 Describe the issues surrounding fitness to stand trial.

In Australia’s criminal justice systems, fitness to stand trial is decided before it can be determined whether a person is responsible for the crime of which he or she is accused. In the Australian legal system, a person is ‘presumed’ to be mentally fit in order to be tried in a court of law. A person can be found by the court to be unfit for trial, on a temporary or permanent basis. If the unfitness is deemed temporary, the proceedings for the offence are stayed until a tribunal, in Queensland the Mental Health Review Tribunal, reviews the matter and decides the person is fit for trial. If the Court finds a person to be permanently unfit for trial, the proceedings for the offence end.

### 13.3 Delineate the conditions under which a person can be committed to a hospital under ‘civil commitment’, and the issues surrounding the rights to receive and refuse treatment.

A person can be civilly committed to an authorised mental health service or public hospital against his or her wishes if the person is mentally ill and presents a risk to their own or others’ safety or welfare. A person can be detained in such facilities for up to 12 hours while the emergency examination authority is being made, to determine the treatment and care needed, or to make a recommendation for assessment. A recommendation for assessment can then be made that allows a person to be sent to an authorised mental health service and detained for some period of time, ranging from 24 hours to 72 hours. Detainment beyond this period requires the authorised doctor to make a treatment authority. The treatment authority authorises the involuntary treatment and care of a person for mental illness. People with psychological disorders who do not abuse substances are not necessarily more likely to engage in violence than are non-mentally ill people who do not abuse substances.

Early studies on the prediction of violence had a number of flaws. Later research has shown that violence can be more accurately predicted if any of these conditions apply: repeated acts of violence, a single serious violent act, being on the brink of violence or medication non-compliance.

Court cases have tried to balance a person’s rights with the rights of society to be protected. The least restrictive alternative to freedom is to be provided when treating people with mental disorders and protecting them from harming themselves and others. A series of court cases have generally supported the notion that those people detained or committed to a hospital have the right to receive

treatment. People with psychological disorders have the right to refuse treatment as well, except when doing so poses a danger to self or others.

Beginning in the 1980s, large numbers of people were released from ‘mental hospitals’ in what has been called deinstitutionalisation. Unfortunately, not enough treatment options are available in the community. Prisons and immigration centres are now the new ‘hospitals’ for people with psychological disorders. Police officers are called on to do the work once reserved for mental health professionals. Partnerships among police, courts and community mental health providers are promising for helping people with psychological disorders.

#### 13.4 Describe the ethics surrounding psychological research and therapy.

Ethical restraints on research are necessary to avoid the abuses that have occurred in the past. Since the *Nuremberg Codes* of 1947, a number of ethical codes regarding psychological research have been developed. Research must be approved for safety and ethics by an institutional review board.

Special precautions must be taken to ensure that research participants with psychological disorders or diminished capacity fully understand the risks and benefits of any research they are asked to participate in. In addition, particular care must be taken to make certain that they can decline or withdraw from research without feeling coerced. Informed consent procedures must include enough information about the research so that participants know about the risks and feel free to withdraw without penalty.

In therapy sessions, people have the right to have what is discussed kept confidential (in other words, it can’t be disclosed to a third party). However, confidentiality can be broken if a person is a danger to self or others, where there is legal obligation to do so, or when consulting colleagues or in the course of supervision or professional training.

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## KEY TERMS

**civil commitment** used in a general sense to refer to the involuntary detention, treatment and care of a person who has, or is reasonably suspected of having, a mental illness and who is considered to be a risk to themselves or others

**confidentiality** a principle observed by lawyers, doctors, pastors, psychologists and psychiatrists that dictates that the contents of a professional and private relationship not be divulged to anyone else

**criminal commitment** a term used in this chapter to describe, in a general sense, the procedure by which a court detains a person involuntarily in a mental health facility or forensic hospital based on the person’s unfitness for trial or unsoundness of mind

**diminished responsibility** abnormality of mind that is found to substantially impair one or more capacities in the insanity defence

**fitness to stand trial** whether an accused has sufficient mental or intellectual capacity to understand the proceedings and to make an adequate defence

**forensic orders** made by the Court if, because of the person’s mental condition, it considers it necessary to protect the safety of the community from the risk of serious harm to other persons or property

**information notice** relates to a patient of an authorised mental health service who is subject to a forensic order or treatment support order. It may contain information that is considered relevant to the safety of victims such as when a forensic patient is granted community leave

**informed consent** the agreement of a person to serve as a research participant or to enter therapy after being told the possible outcomes, both benefits and risks

**insanity defence** the defence that a disordered mind cannot be a guilty mind and only a guilty mind can engender culpable actions; has three capacities: whether the person knew the nature and quality of the conduct; or that the conduct was wrong; or whether the person was able to control their actions

**involuntary detention** detention that can involve deprivation of a person's liberty, their compulsory treatment (medication) and placement in a mental health service or other authorised place (public hospital)

**least restrictive alternative** to be provided when treating people with psychological disorders and protecting them from harming themselves and others. In general terms, mental health professionals have to provide the treatment that restricts the person's liberty to the least possible degree while remaining workable

**Mental Health Review Tribunal** a specialist body that reviews the involuntary detention and treatment of patients

**mental impairment** in some states and jurisdictions, this is the successor to the insanity defence

**M'Naghten rule** formulated in the aftermath of a murder trial in England in 1843, has since been typically presented to juries as the standard used to determine whether an accused person, who raises the insanity defence, knew the difference between right and wrong at the time of doing the unlawful act

**treatment support order** an order made if the Court considers a person poses a serious risk of harm to others or their property and needs treatment and care for a mental illness

**unsound mind** a state of mental disease of natural mental infirmity as described in s27 of the Criminal Code

**victim impact statement** a statement that may be prepared by a victim or a close relative of a victim (e.g., spouses, siblings, children, parents and extended family members) and given to the prosecuting authority to present to the Mental Health Court when it is determining a person's soundness of mind or fitness to stand trial (s162). Alternatively, a victim impact statement can be presented to the Mental Health Tribunal on review of a forensic order or treatment support order (s742)

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## WEBSITES

The *Conversation* articles suggested below are written by academics and researchers in psychology and discuss important issues related to individuals living with mental illnesses within our society.

1. 'Are you really at risk of attack by someone with schizophrenia?' (<https://theconversation.com/are-you-really-at-risk-of-attack-by-someone-with-schizophrenia-14656>)
2. 'Violence and mental illness: harsh reality demands sensitive answers' (<https://theconversation.com/violence-and-mental-illness-harsh-reality-demands-sensitive-answers-23460>)

The following websites provide access to the Australian and New Zealand Code of Ethics and all other important ethical guidelines required when practising as a psychologist.

3. APS Code of Ethics ([www.psychology.org.au/Assets/Files/APS-Code-of-Ethics.pdf](http://www.psychology.org.au/Assets/Files/APS-Code-of-Ethics.pdf))
4. Code of Ethics for Psychologists Working in Aotearoa/New Zealand ([www.psychology.org.nz/wp-content/uploads/2014/04/code-of-ethics.pdf](http://www.psychology.org.nz/wp-content/uploads/2014/04/code-of-ethics.pdf))

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## REFERENCES

- Appelbaum, P. S., & Gutheil, T. (1991). *Clinical handbook of psychiatry and the law*. Baltimore: Williams & Wilkins.
- Australian Psychological Society (APS) (2007). *Code of ethics*. Melbourne, Vic.: Author.
- Beecher, H. K. (1966). Ethics and clinical research. *New England Journal of Medicine*, 274, 1354–1360.
- Bilowol, J., & Davis, B. (2007). Struggling with its massacre in silence. *Sydney Morning Herald*. Retrieved from [www.smh.com.au/news/tasmania/struggling-with-its-massacre-in-silence/2007/02/07/1170524157142.html](http://www.smh.com.au/news/tasmania/struggling-with-its-massacre-in-silence/2007/02/07/1170524157142.html)
- Blake, S. (2015, September 13). Monster on the inside. *The Daily Telegraph*. Retrieved from [www.dailytelegraph.com.au/news/special-features/in-depth/monster-on-the-inside/news-story/751e16b4517c9d4d4226e6ba78d5b079](http://www.dailytelegraph.com.au/news/special-features/in-depth/monster-on-the-inside/news-story/751e16b4517c9d4d4226e6ba78d5b079)
- Campbell, J., Stefan, S., & Loder, A. (1994). Putting violence in context. *Hospital and Community Psychiatry*, 45, 633.
- Capron, A. M. (1999). Ethical and human rights issues in research on mental disorders that may affect decision-making capacity. *New England Journal of Medicine*, 340, 1430–1434.
- Caxton Legal Centre. (2011). *The Queensland law handbook: Your practical guide to the law* (11th ed.). South Brisbane: Caxton Legal Centre.

- Carpenter, W. T., Gold, J. M., Lahti, A. C., Queern, C. A., Conley, R. R., Bartko, J. J., ... Appelbaum, P. S. (2000). Decisional capacity for informed consent in schizophrenia research. *Archives of General Psychiatry*, 57, 533–538.
- Corrigan, P. W., & Watson, A. C. (2005). Findings from the National Comorbidity Survey on the frequency of violent behavior in individuals with psychiatric disorders. *Psychiatry Research*, 136, 153–162.
- Crowley-Cyr, L. (2005). Contractualism, exclusion and ‘madness’ in Australia’s ‘outsourced wastelands’. *Macquarie Law Journal*, 5, 81–102.
- Crowley-Cyr, L. (2010). *Homelessness, mental illness and the State: an endless crisis of suffering and exclusion*. Saarbrücken, Germany: VDM Verlag Dr Muller.
- Dautremont v Broadlawns Hospital, 827 F.2d 291 (8th Cir. 1987)
- Department of Health and Ageing (2004). *National Mental Health Report 2004: Eighth Report. Summary of Changes in Australia’s Mental Health Services under the National Mental Health Strategy 1993–2002*. Canberra: Commonwealth of Australia.
- Douglas, K. S., Guy, L. S., & Hart, S. D. (2009). Psychosis as a risk factor for violence to others: a meta-analysis. *Psychological Bulletin*, 135, 679–706.
- Ewing v Goldstein, Cal. App. 4th B163112.2d. (2004)
- Fazel, S., Gulati, G., Linsell, L., Geddes, J. R., & Grann, M. (2009). Schizophrenia and violence: Systematic review and meta-analysis. *PLoS Medicine*. [www.plosmedicine.org/article/info%3Adoi%2F10.1371%2Fjournal.pmed.1000120](http://www.plosmedicine.org/article/info%3Adoi%2F10.1371%2Fjournal.pmed.1000120)
- Furniss v Fitchett [1958]NZLR 396
- Grisso, T., Davis, J., Vesselinov, R., Appelbaum, P. S. & Monahan, J. (2000). Violent thoughts and violent behavior following hospitalization for mental disorder. *Journal of Consulting and Clinical Psychology*, 68, 388–398.
- Hedlund v Superior Court, 34 Cal.3d 695 (1983)
- Hemming, A. (2008). It’s time to abolish diminished responsibility, the coach and horses’ defence through criminal responsibility for murder. *University of Notre Dame Australia Law Review*, 10.
- Homicide Act 1957 s2(1)
- Human Rights and Equal Opportunity Commission. (1993). *Human rights and mental illness: Report of the National Inquiry into Human Rights of People with Mental Illness*. Canberra: Australian Government Publishing Service.
- Jablonski by Pahls v United States, 712 F.2d 391 (1983)
- Kiesler, C. A. (1991). Changes in general hospital psychiatric care. *American Psychologist*, 46, 416–421.
- Kozol, H., Boucher, R., & Garofalo, R. (1972). The diagnosis and treatment of dangerousness. *Crime and Delinquency*, 18, 37–92.
- MacKenzie R. D., McEwan T. E., Pathé M. T., James D. V., Ogloff J. R. P., Mullen P. E. (2009). *Stalking risk profile: Guidelines for the assessment and management of stalkers* (1st ed.). Melbourne, Victoria, Australia: StalkInc. and Centre for Forensic Behavioural Science, Monash University.
- Marie, D., & Miles, B. (2008). Social distance and perceived dangerousness across four diagnostic categories of mental disorder. *Australian and New Zealand Journal of Psychiatry*, 42, 126–133.
- Marson, D. C. (2001). Loss of competency in Alzheimer’s disease: Conceptual and psychometric approaches. *International Journal of Law and Psychiatry*, 24, 267–283.
- Marson, D. C., Huthwaite, J. S., & Hebert, K. (2004). Testamentary capacity and undue influence in the elderly: A jurisprudent therapy perspective. *Law and Psychology Review*, 28, 71–96.
- McIlwraith, J., & Madden, B. (2014) *Health care and the law, sixth edition*. Sydney: Thomson Reuters Lawbook.
- Ministry of Health. (2012). *Rising to the challenge: The Mental Health and Addiction Service Development Plan 2012–2017*. Wellington: Ministry of Health.
- M’Naghten’s Case. (n.d.). *Casebriefs.com*. Retrieved from [www.casebriefs.com/blog/law/criminal-law/criminal-law-keyed-to-kadish/exculpation/mnaghtens-case/](http://www.casebriefs.com/blog/law/criminal-law/criminal-law-keyed-to-kadish/exculpation/mnaghtens-case/)
- Monahan, J. (1973). The psychiatrization of criminal behavior. *Hospital and Community Psychiatry*, 24, 105–107.
- Monahan, J. (1976). The prevention of violence. In J. Monahan (Ed.), *Community mental health and the criminal justice system*. Elmsford, NY: Pergamon Press.
- Monahan, J. (1984). The prediction of violent behavior: Toward a second generation of theory and policy. *American Journal of Psychiatry*, 141, 10–15.
- Monahan, J., & Steadman, H. (1994). Toward a rejuvenation of risk assessment research. In J. Monahan & H. Steadman (Eds.), *Violence and mental disorder: Developments in risk assessment*. Chicago: University of Chicago Press.
- Morse, S. J. (1992). The “guilty mind”: Mens rea. In D. K. Kagehiro & W. S. Laufer (Eds.), *Handbook of psychology and law* (pp. 207–229). New York: Springer-Verlag.
- New Zealand Psychological Society (2002). *Code of ethics*. Retrieved from [www.psychology.org.nz](http://www.psychology.org.nz).
- NHMRC (2007). *National statement on ethical conduct in human research (2007) — Updated December 2013* (the National Statement). Commonwealth of Australia, Canberra.
- Nielssen, O. B., Bourget, D., Laajasalo, T., Liem, M., Labelle, A., Hakkanen-Nyholm, H., ... & Lange, M. M. (2011). Homicide of strangers by people with a psychotic illness. *Schizophrenia Bulletin*, 37, 572–579.
- Nielssen, O. B., Westmore, B. D., Large, M. M., & Hayes, R. A. (2002). Homicide during psychotic illness in New South Wales between 1993 and 2002. *The Medical Journal of Australia*, 186, 301–304.

- Ogloff, J. R., & Daffern, M. (2006). The dynamic appraisal of situational aggression: An instrument to assess risk for imminent aggression in psychiatric inpatients. *Behavioural Science Law*, 24(6), 799–813.
- Peck v Counseling Service of Addison County, 499 A.2d 422 (Vt. 1985)
- Quinsey, V. L., Harris, G. T., Rice, M. E., & Cormier, C. A. (2006). *Violent offenders: Appraising and managing risk* (2nd ed.). Washington, DC: American Psychological Association.
- R v Fitchett [2010]VSC 393
- R v Freeman [2011]VSC 139
- R v M’Naghten 8 ER 718, [1843]UKHL J16
- R v Porter (1933) 55 CLR 182.
- R v Pritchard (1836) 173 ER [304].
- R v Presser (1958) 45 VR.
- Robinson, D. N. (1996). *Wild beasts and idle humours: The insanity defense from antiquity to the present*. Cambridge, Massachusetts: Harvard University Press.
- Ruiz v Johnson, 37 F. Supp. 2d 855 (S.D. Texas 1999)
- Ryan, C. (2016, August 31). Should we be forcing people with severe mental illness to have treatment they don’t want? *The Conversation*. Retrieved from <https://theconversation.com/should-we-be-forcing-people-with-severe-mental-illness-to-have-treatment-they-dont-want-63762>
- Schwitzgebel, R. L., & Schwitzgebel, R. K. (1980). *Law and psychological practice*. New York: John Wiley & Sons.
- Shelter NSW. (2002). *Submission to the NSW Legislative Council Select Committee on Mental Health Inquiry into and Report on Mental Health Services in NSW*. Retrieved from <https://shelternsw.org.au/sites/shelternsw.org.au/files/public/documents/sub02mentalhealthinquiry.pdf>
- Shepherd, S. M., & Strand, S. (2016). The PCL: YV and re-offending across ethnic groups. *Journal of Criminal Psychology*, 6(2), 51–62.
- Skeem, J. L., & Monahan, J. (2011). Current directions in violence risk assessment. *Current Directions in Psychological Science*, 20, 38–42.
- Smith v Pennington [2015]NSWSC 1168
- Steadman, H. J., Mulvey, E. P., Monahan, J., Robbins, P. C., Appelbaum, P. S., Grisso, T., Roth, L. H., & Silver, E. (1998). Violence by people discharged from acute psychiatric inpatient facilities and by others in the same neighborhoods. *Archives of General Psychiatry*, 55, 393–401.
- Stewart, J. (2016). The man who spent 42 years in jail as one of Australia’s longest serving prisoners. *Lateline*. Retrieved from [www.abc.net.au/lateline/content/2015/s4405918.htm](http://www.abc.net.au/lateline/content/2015/s4405918.htm)
- Swanson, J. W., Holzer, C. E., Ganju, V. K., & Jono, R. T. (1990). Violence and psychiatric disorder in the community: Evidence from the Epidemiological Catchment Area surveys. *Hospital and Community Psychiatry*, 41, 761–770.
- Tarasoff v Regents of the University of California, 529 P.2d 553 (Cal. 1974), vacated, reheard in bank and affirmed, 131 Cal. Rptr. 14, 551 P.2d 334 (1976). The 1976 California Supreme Court ruling was by a four-to-three majority.
- Thomas S v Flaherty, 902 F.2d 250, cert. denied, 111 S.Ct. 373 (1990).
- Torrey, E. F. (2014). *American psychosis: How the federal government destroyed the mental illness treatment system*. Oxford, UK: Oxford University Press.
- United States v Charters, 863, F.2d 302
- Veen v R (No 2) [1988]HCA 14
- W v Edgell [1990]1 All ER 855 (CA England)
- Webster, C., Douglas, K., Eaves, D., & Hart, S. (1997). *HCR-20: Assessing risk for violence (Version 2)*. Vancouver, British Columbia, Canada: Simon Fraser University.
- Wirshing, D. W., Wirshing, W. C., Marder, S. R., Liberman, R. P., & Mintz, J. (1998). Informed consent: Assessment of comprehension. *American Journal of Psychiatry*, 155, 1508–1511.
- Wyatt v Stickney, 325 F.Supp. 781 (M.D. Ala. 1971), enforced in 334 F.Supp. 1341 (M.D. Ala. 1971), 344 F.Supp. 373, 379 (M.D. Ala.1972), *aff’d sub nom Wyatt v Anderholt*, 503 F.2d 1305 (5th Cir. 1974).

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